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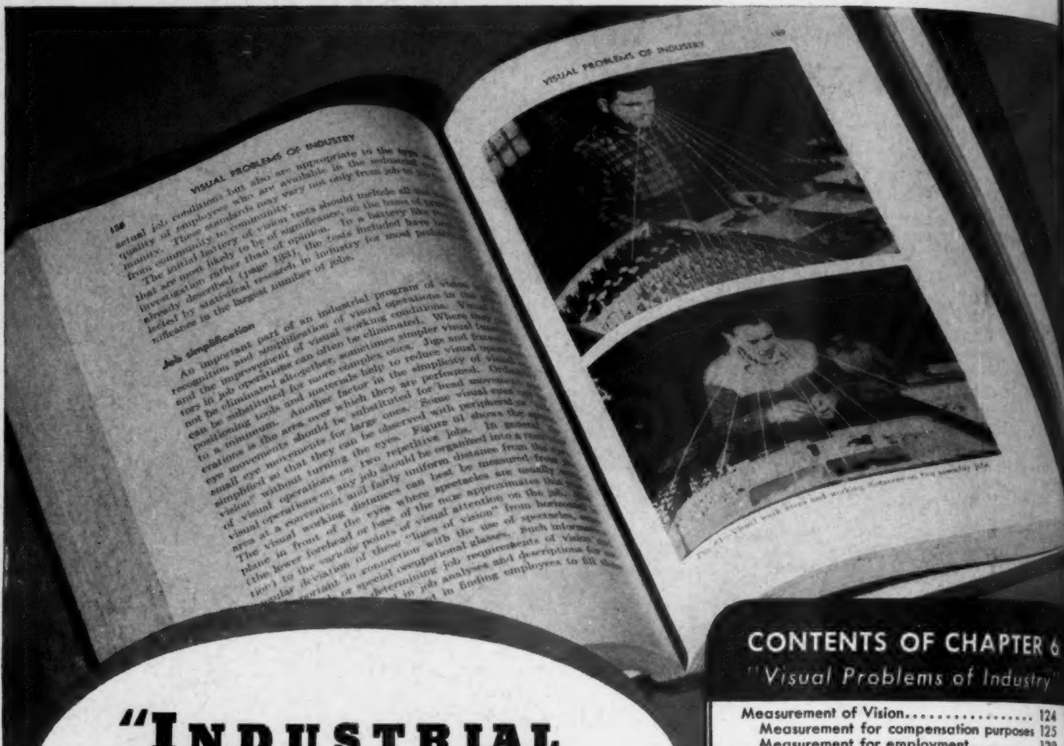

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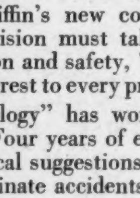

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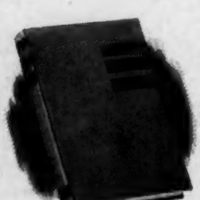

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Walter B. Lancaster

FOREWORD

A tribute to Dr. Walter B. Lancaster on his eightieth birthday was proposed to a group of his devoted colleagues and pupils at the Atlantic City meeting of the American Medical Association by Dr. James J. Regan of Boston. That Dr. Lancaster in his eightieth year still maintained his leadership and productivity in ophthalmology, appealed instantly as affording an almost unique opportunity. Perhaps the last professional act of Dr. Edward Jackson and Dr. Allen Greenwood was the delighted approval of a birthday number of the American Journal of Ophthalmology dedicated to Dr. Lancaster. Dr. Derrick Vail agreed. Dr. Greenwood as chairman immediately issued invitations to his colleagues to support the project. Heartened by the vigorous response, a committee met at the subsequent meeting in Chicago of the American Academy of Ophthalmology and Otolaryngology. It included an officer of the Academy, Dr. Ralph I. Lloyd, and of the American Ophthalmological Society, Dr. John Green. The plan was received with enthusiasm by the editors of the Journal, but they more than doubted the possibility of issuing such a periodical in the brief time before the anniversary. Fortunately, the committee, in its inexperience, was blissfully unaware of the obstacles and proceeded to gather material.

Instead of having to ask for contributions, the committee found themselves put to it to find space, under war conditions, for the important material offered as a tribute to Dr. Lancaster. It is gratifying that many of these fresh and important contributions report studies which were either inspired or influenced by Dr. Lancaster. This is the more significant when it is realized that pressure of time limited the selection to material already in shape for publication. The broad scope—operative, clinical, and scientific—of the studies which he has encouraged is indicative of his versatility and his impress upon the profession.

Only the punctuality of the contributors and the unremitting efforts of the Journal staff have made possible the prompt publication. For this the committee is sincerely grateful. They wish particularly to thank Miss Emma S. Buss, the manuscript editor, and Miss Helen A. Denehy and Mrs. Margot Y. Lusk, secretaries to Dr. Lancaster, who compiled the bibliography and furnished biographic data. To Dr. Lawrence T. Post, acting editor-in-chief of the Journal, whose unstinted help and advice made the issue possible, they offer special thanks. The ultimate success, as well as the inspiration, of the enterprise is due to the loyal and indefatigable secretary, Dr. James J. Regan. Circumstances have often necessitated his making decisions and arrangements in which he would ordinarily have had assistance from the rest of the committee. All have,

however, delighted in the privilege of participating in this tribute to our distinguished and revered teacher and friend, Dr. Lancaster.

For the Committee, S. Judd Beach, M.D.

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 26 MAY, 1943 NUMBER 5

WALTER B. LANCASTER, M.D.

A BIOGRAPHICAL SKETCH AND BIBLIOGRAPHY 1897-1943

JAMES J. REGAN, M.D.
Boston

Walter Brackett Lancaster was born on May 11, 1863, in Newton, Massachusetts. On both sides his family traced back to the Mayflower, but numbered no very distinguished names. After attending the Newton schools, he entered Harvard at the age of 17 years. Entering the Harvard Medical School in 1884, he should have been graduated in 1887, but was out two years on account of ill health.

In December, 1885, he married Emma Winter. They have one child Miss Julia E. Lancaster, orthoptist in San Francisco.

After receiving his degree from Harvard Medical School in 1889, he went at once to Europe. In Vienna, he had the good fortune to study under Professor Mauthner, who had recently moved to that city from Innsbruck. Mauthner was master in the fields of refraction, motility, and neuro-ophthalmology. During much of the time Lancaster was his only pupil. From Vienna Lancaster went to Paris and then to Edinburgh, where Maddox was also a student; and finally he spent a few weeks in London.

On returning to the United States, he began general practice in Wellesley Hills. After a few months, he opened an office in Boston for ophthalmic practice and joined the staff of the Boston City Hospital. As soon as his practice warranted, he gave up general practice and devoted himself to ophthalmology. He then joined the staff of the Massachusetts Eye and Ear Infirmary.

Invited to offer courses of instruction in the Boston Polyclinic, he gave courses in clinical ophthalmology, in refraction, and in operative surgery, a novelty at that time. Later the Polyclinic was closed and graduate teaching begun by Harvard Medical School. Lancaster was one of the large staff of assistants in ophthalmology.

In 1901, Lancaster's health became worse and he retired from practice and left Boston. After a few years, he was able to take special work at the Massachusetts Agricultural College at Amherst, as he planned to live on a farm.

By 1908, he was enough better so that his doctor suggested that he start practicing in Amherst. As practice increased, he again found his health failing, and he decided to spend a year in Europe for a complete vacation.

While in England, he consulted Sir Berkley Moynihan, who advised operation for duodenal ulcer. This had been Lancaster's own diagnosis but had not been concurred in by his Boston friends. Operation revealed no duodenal ulcer but a bad, old, chronic, adherent appendix. Following this operation, improvement was rapid.

On returning to America, he was able to reopen an office in Boston and resume hospital work and teaching. Looking over the field, it seemed to him that the weak spot in ophthalmic teaching was physiologic optics. He offered to give a graduate course at the Harvard Medical School

and was assigned a room but no funds. He raised a few hundred dollars; enough to set up simple laboratory apparatus.

Some of his friends said "Who is going to take your course?" Lancaster had not thought of that—he had assumed that a course in such a needed subject would be eagerly sought. On reflection, it occurred to him that if an examination could be set up, which all men must take who wished to show that they were qualified to practice ophthalmology, the subject of physiologic optics would be required and so a course on that subject would be very much needed.

It was at this time that Dr. Jackson was writing and speaking on improving the teaching of ophthalmology. During the next year or two, Dr. Jackson would read a paper and Dr. Lancaster would discuss it and then Dr. Lancaster would read a paper and Dr. Jackson would discuss it. This must have happened once too often, for the last paper read by Dr. Lancaster before the American Ophthalmological Society, which was discussed by Dr. Jackson, was never printed in the Transactions.

In 1915, the three Societies all voted to set up a joint board for ophthalmic examinations. Lancaster was a member of this original board and of the Committee on Examinations. As the junior member, he acted as secretary of this subcommittee and drew up the plans for the examinations, which were adopted, with little change, and have been followed by most of the dozen or more specialty boards since formed.

The laboratory of physiologic optics at Harvard was carried on for a few years with a few pupils and some research. The chief item of research was the discovery of the viscosity of the lens, due to which the effect of continuous strong accommodation is, in addition to the first rapid (fraction of a second) increase in the refractive power, a slow (5 to 15 minutes) continuous *further increase* of 25 to 50 percent more refractive power (British Journal of Ophthalmology, 1914).

As the Medical School had no funds which it wished to devote to this laboratory, Lancaster gave it up. He did this more cheerfully, as his practice was becoming more time-consuming.

About this time, Dr. Walter Pyle of Philadelphia, editor of an international set of monographs on ophthalmology by such men as Darier, Collins and Mayou, Knapp, and others, asked Lancaster to write the volume on operative surgery. This appealed to Lancaster, who was much interested in the subject, having inaugurated the teaching of operative ophthalmic surgery in Boston. Several of the chapters were published in the journals. Then came the war, and the publishers released Lancaster from his contract, which called for completion of the book the next year.

Lancaster's first war service was at Camp Devens. When Dr. Wilmer and Dr. Berens and their associates left the Research Laboratory of the Air Service at Mineola for the work in France, Lancaster was offered the appointment as chief of the ophthalmic section which Wilmer had vacated. He had to choose between this, and the job of teaching ophthalmology at Chattanooga to the junior ophthalmologists in the Army, or a job as commanding officer of a Camp. He chose the Research Laboratory. Of the pieces of research he set in motion there, the one that has become widely known is the work on stereoscopic depth perception, which he assigned to Captain Howard. It was Lancaster's conviction that good stereopsis was less important for flyers than was generally claimed, due to the fact that it is effective only for objects relatively near together and relatively near

the observer. The task laid out for Howard consisted first of selected references to the literature to provide proper background. Then some experiments on depth perception out of doors at distances of 50, 100, and 300 meters. Howard became interested in the apparatus devised for measuring stereopsis, and before he had completed his work on this preliminary matter, the armistice came. The modification of the apparatus which he produced, with the help of the instrument maker, was brought out after the armistice. Lancaster apparently still holds his opinion that stereopsis is of secondary importance in flying (see discussion of paper by Verhoeff describing a new test for stereopsis, Archives of Ophthalmology, 1942, December).

Three months after his appointment, warning came from Washington, where they could see that the end was approaching, to hold up plans for the Research Laboratory on which Lancaster was working, and so, little came of his term of service there.

In 1926, Dr. Lancaster resigned from active service at the Eye and Ear Infirmary and from the Harvard Graduate Medical School, not wishing to wait for the automatic retiring age. Ten years later, he was asked to join the Harvard teaching staff and has continued as lecturer in ophthalmology ever since.

In 1929, he invited Professor Ames of Dartmouth to meet Professor Bielschowsky of Breslau at his office, hoping that they might form a working alliance. This succeeded, and Professor Bielschowsky spent the remaining years of his life at Hanover, as Director of the Dartmouth Eye Institute.

After Dr. Bielschowsky's death, Dr. Lancaster was invited to the Dartmouth Eye Institute. He was made chief of staff, but not director of the Institute, and served two years. The Institute is governed by a board of directors. Their ideals of what the Dartmouth Eye Institute should aim to be were irreconcilable with Dr. Lancaster's ideals, and so he returned to private practice.

Dr. Lancaster received the following degrees: Harvard, A.B. magna cum laude 1884; Harvard, M.D. 1889; Dartmouth, D.Sc. Honoris Causa 1939.*

He has been president of the New England Ophthalmological Society; of the American Academy of Ophthalmology and Otolaryngology; chairman of the Section on Ophthalmology of the American Medical Association; president of the American Ophthalmological Society; member of the Council of the Association for Research in Ophthalmology, American College of Surgeons, the Il-

*Upon conferring this degree Dr. Ernest Martin Hopkins made the following citation:

"Walter Brackett Lancaster: Graduate of the College and of the Medical School of Harvard University; now after half a century of distinguished practice in your delicate specialty you are, in the universal admiration of your colleagues but with characteristic modesty, dean of American ophthalmology. Endowed with rare skill as a surgeon; versed as few are in the theory and practice of treatment of the eye; always in the forefront in creating, encouraging, and utilizing new developments in the field, your own scientific papers over the last four decades in number and scope in themselves constitute an ophthalmological library of admirable stature. Ever alert to potential new departures of importance in your field of specialization, your interest in physiological optics was an indispensable impetus to the original establishment of the Department of Research in Physiological Optics at Dartmouth. Not only do patients in great numbers who have been the grateful beneficiaries of your skilled ministrations look to you as protector of the most precious of human senses, but many times that number, the healing of whose eyes has been aided by procedures wholly or in part attributable to you, are unknowingly in the debt of one whose life has been devoted to the aim that for human beings light shall not fail. Upon you as one to whom multitudes are under obligation and to whose friendly interest and approval this college is especially indebted, I confer the honorary degree of Doctor of Science."

luminating Engineering Society, and of its Committee on Research, Optical Society of America and one of the editors of its Journal; member of the National Conference on Nomenclature of Disease and author (with the aid of several associates) of section xi, Diseases of the eye, in which all diseases of the eye are classified anatomically and etiologically —one of his major tasks—member of the Committee on Scientific papers of the International Congress of Ophthalmology held at Washington, D.C., in 1923; one of the original members of the American Board of Ophthalmology, several times president, member of the executive committee of the Advisory Board of Medical Specialties; member of the American Association for Advancement of Science.

His hospital appointments were: Ophthalmic Surgeon, Boston City Hospital (Senior at the time of his resignation) now Consulting Ophthalmic Surgeon; Ophthalmic Surgeon at the Massachusetts Eye and Ear Infirmary, now Consulting Ophthalmic Surgeon, ditto Massachusetts General Hospital. He was instrumental in founding the Boston Nursery for Blind Babies and was its first consultant. He was Chief of Staff of the Dartmouth Eye Institute 1940-1942, and Ophthalmic Surgeon of the Mary Hitchcock Memorial Hospital at Hanover at the same time.

In politics independent (usually Republican). In religion Congregationalist, having joined that denomination in 1875, he took his religion seriously. In later years he was identified with the Episcopal denomination to which his wife and daughter belonged.

Lancaster's recreational and social interests were always limited by the demands of his professional career, but he was fond of golf and enjoyed very much playing with his ophthalmic friends at the ophthalmic meetings.

Since the termination of his work at Dartmouth, he has returned to Boston where he has resumed private practice. He is still active at medical meetings, especially of the American Board of Ophthalmology.

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NOTE: A limited number of copies of the frontispiece are available to our readers upon application to the Ophthalmic Publishing Company, 837 Carew Tower, Cincinnati, Ohio.

ANGIOMATOSIS RETINAE (VON HIPPEL'S DISEASE). RESULTS FOLLOWING IRRADIATION OF THREE EYES*

FREDERICK C. CORDES, M.D., AND
OWEN C. DICKSON, M.D.
San Francisco

So few data are available regarding the results of treatment of angiomatosis retinae that the reporting of three eyes, one irradiated and observed over a period of 3½ years, the other two for two years after similar treatment, seems justified.

The case observed 3½ years has been previously reported one year after irradiation.[1] At the time of that presentation a complete survey of the literature was made. In the present paper the emphasis will be placed on the therapeutic measures that have been employed and the results reported. In order to realize that any type of therapy must have a limited place in the treatment of this disease, it is necessary that the entire picture be understood. Consequently a brief résumé seems to be in order.

HISTORY

The first description of angiomatosis retinae in the literature is by E. Fuchs,[2] who, in 1882, described it under the title of "Aneurysma arteriovenosum traumaticum." Following this report the disease was described a number of times, under various titles, until 1904, when von Hippel[3] established angiomatosis retinae as a clinical entity. In 1927 Lindau[4] demonstrated the close association between angiomatosis retinae and the occurrence of angiomatosis and cystic lesions in the central nervous system, usually the cerebellum. He also demonstrated that other viscera—namely, kidneys, pancreas, ovaries, and suprarenal glands—are often involved. Since that time the disease has been known as the von Hippel-Lindau syndrome. Approximately 160 cases of angiomatosis retinae have been reported in the literature.

OCULAR MANIFESTATIONS AND CAUSE OF THE DISEASE

According to Bedell,[5] the earliest photographic sign is fullness of the retinal veins. Ditroi[6] states that the first ophthalmoscopic change is a fan-shaped anastomosis between one branch of the central artery and the central vein, which is the beginning of the tumor formation.

From this rete mirabile a berrylike reddish-colored mass develops that is sharply demarcated from the surrounding area and is supplied by an artery and vein. Characteristically, both vessels develop into wormlike enlarged stems carrying dark blood, and in a short time it is impossible to differentiate between the artery and the vein. The retina surrounding the tumor is slightly elevated. Around the macula and disc shiny white spots of exudate make their appearance, which at first are pinhead in size and later become confluent. At times this exudate is arranged as a stellate figure in the macula. Frequently there are also collections of exudate along the vessels. A little later a globular detachment of the retina appears, surrounding the enlarged tumor mass. In this stage the exudate increases in amount and assumes a yellowish color. The condition progresses until there is a massive detachment of the retina, which protrudes into the vitreous. Glistening yellowish-white spots of exudate are present over the entire fundus during this stage. The

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vessels leading to the tumor become markedly enlarged, the disc becomes atrophic, and the eye amaurotic.

It is noteworthy that frequently these vascular tumors are multiple either in the beginning or later in the course of the disease. They retain their globular appearance only in the early stages and later become more tumorlike, with nobs and irregularities. The white or yellowish white circumscribed areas of exudate may be extensive over the retina and may produce a picture similar to that of retinitis circinata or retinitis albuminurica. These areas of degeneration, however, are usually slightly elevated, a condition not found in the previously mentioned retinitis. In addition, they are often surrounded by a ring of dark red due to the fact that the proliferating glial tissue has absorbed some pigment from the degenerating pigment layer. Hemorrhages may also occur in the retina and vitreous and at times may be severe. Detachment of the retina is always present in cases in which the condition is advanced, usually developing from the periphery and giving the impression of being semisolid. In some instances, transillumination gives the impression of a solid detachment and has led to the diagnosis of retinoblastoma or other intraocular tumors. The complete detachment of the retina which often has the appearance as though it lay over, or was permeated by, coagulated milk, completes the picture in the late stages. Finally iridocyclitis develops, complicated by secondary glaucoma and eventual opacification of the lens.

Ocular microscopic pathologic picture

To consider therapy intelligently one must be familiar with the microscopic pathologic picture.

Microscopically, the growth is composed of a plexus of thin-walled capillary blood vessels situated in the retina. At times there may be small solid masses of endothelial cells. The cells between the vessels show few mitoses, whereas the feeding vessels are greatly dilated and thickened. In certain areas the growth tends to undergo cystic degeneration. Lying between the vessels are curious fat-laden cells, similar to the pseudoxanthoma cells seen in hypernephroma. The fat is dissolved out in the usual preparation of the microscopic section, so that the cytoplasm has a honeycomb appearance. The vessels have a tendency to give rise to hemorrhage in the retina, vitreous, or subretinal space. In the case of hemorrhage in the vitreous, newly formed blood vessels often extend into the coagulum, with the subsequent development of fibrous tissue the later contraction of which is probably a factor in the production of retinal detachment. Subretinal hemorrhage may organize into hard fibrous tissue containing cholesterol crystals, which ophthalmoscopically gives the appearance of white plaques with retinal vessels running over them. In the later stages the glial proliferation is a prominent feature in the picture. This is nicely demonstrated in the work of McDonald and Lippincott;[7] Collier[8] emphasized the importance of remembering that the retinal angioma may be microscopic in size.

Age and sex incidence

Most of the cases of angiomatosis retinae have occurred in growing adults. The average age of the patient, according to Lindau,[9] is 25 years. The youngest patient of whom such a case was reported was a male infant aged two years, whereas the oldest was a woman of 50 years. Both eyes were involved in about 50 percent of the patients. One third of the eyes involved had multiple tumors; Lindau[10]

reported cerebral involvement in 25 percent of the cases, these cerebral signs on the average appearing 10 years after the discovery of the ocular lesions.

Collier[8] reported 20 percent of his series showing familial incidence and stated that it is usually transmitted through the female, although 60 percent of the cases are reported in males.

Prognosis

In angiomatosis retinae the prognosis is poor not only as to the first eye, but also as far as the second eye is concerned, for the disease is bilateral in 50 percent of cases. At first the visual acuity is good, but later the retina undergoes degeneration, followed by complete detachment, in most instances associated with secondary glaucoma. Changes in the blood vessels take place over a wide range. The rate of progression may vary a great deal. Rumbaur[11] believes that the earlier the disease process appears the more rapidly progressive is the course, and that the manifestations are more marked and widespread in the third generation of an affected strain. He also believes that there may be an occasional spontaneous healing of the retinal lesions.

Therapy

Most of the cases of angiomatosis retinae occur in early adult life, and in the 25 percent of patients who develop Lindau's disease the symptoms of the latter do not appear for an average of 10 years later. Inasmuch as the prognosis for vision is bad and as 50 percent of the cases become bilateral, any therapeutic measure that offers hope of conserving the sight warrants consideration.

A survey of the literature shows surprisingly few data regarding treatment of angiomatosis retinae.

Before the disease was understood, intravenous injections of arsphenamine, tuberculin, calcium gluconate, and foreign-protein therapy were used unsuccessfully.

In recent years attempts at therapy have been limited to electrolysis, diathermy, and irradiation with radium and X rays.

E. T. Collins[12] in 1930 suggested electrolysis as a possible therapy and suggested that localization of the tumor be effected according to the methods employed by Gonin. Neame[13] treated an early case by electrolysis, using a special needle similar to that employed in the treatment of spider nevi of the skin. The effect was noticeable in the mottled pigmentation in the neighborhood of the angiomas. No report has been made as to the final outcome.

Diathermy has been used a number of times. Weve[14] reported the production of scar tissue and preservation of vision of 1/6 two-and-a-half years following diathermy puncture at the site of a lesion. He stated that in early lesions surface diathermy might be adequate but suggested severing of vessels supplying the tumor by diathermy puncture in the event of large lesions. Kaye[15] reported two cases treated by Stallard, who used surface and puncture diathermy. Twelve months after treatment the involved areas resembled sites of retinal-detachment operations, but the tumor growth had been halted. Rumbaur[11] used repeated diathermy puncture over the site of a lesion, with preservation of vision of 4/60 over a six-months period. On October 13, 1942, at a meeting of the American Academy of Ophthalmology and Otolaryngology, P. W. Lewis reported a case in which he used diathermy punctures, to the destruction of the angioma. The case was followed for a year.

Radium has been used successfully a number of times. Holm[16] employed it in two cases and witnessed shrinking of the tumors, but did not record the end results.

R. Foster Moore[17] reported two cases in which treatment with radium was successful. Both patients, women aged 24 and 36 years, had previously lost one eye due to the disease. In the first patient's eye he placed three 4-millicurie seeds of radon as near the growth as possible. These seeds were left *in situ* for 10 days. A good deal of reaction occurred, which gradually subsided. Six months later there was no neoplasm, the site being occupied by pale stippled pigmented areas. The vessels supplying the growth had become obliterated. In the second case a much smaller dose of seeds was used—namely, two seeds of 2 millicuries each, which were in place only three days. At the end of three months only scar tissue was present at the site of the angiomatosis. This patient had a large subhyaloid hemorrhage two years later and lost the sight of the eye. Traquair[18] applied 80 mg. of radium for 12½ hours over an angiomatosis-retinae lesion of a woman, aged 38 years. Two-and-a-half months later 15 mg. was applied subconjunctivally for 72 hours. Sixteen months later the vision was 6/12 and examination of the fundus showed the vessels to be smaller and the mass more fibrous. McDonald and Lippincott[7] applied radium unsuccessfully in a case in which the condition was well advanced. X-ray therapy in angiomatosis retinae has been reported a number of times.

Mulock Houwer[19] found roentgen therapy unsuccessful in an advanced case in which there were changes in the vitreous and detachment of the retina preceding treatment. Erggelet's[20] case also was well advanced and showed no improvement. There are other similar reports of unsuccessful results in well-advanced cases, which can be readily understood when one remembers the pathology of the later stage of angiomatosis retinae.

Craig, Wagener, and Kernohan[21] reported a case in which three courses of X-ray therapy were given to the eye. Three years following this irradiation the large vessels leading to the mass persisted, but the angioma itself seemed to consist for the most part of scar tissue associated with atrophy and a degenerated condition of the surrounding retina. Vision of the eye three years after irradiation was 6/10.

Case reports

Case 1

The history of this case, which has been reported in detail,[1] will be resumed for the period covering the first year after irradiation.

R. M. P., a 20-year-old, single girl, came to the Ophthalmic Clinic of the University of California for examination, on August 11, 1938.

Past history and family history were essentially negative.

General physical, neurologic, and roentgen examinations were negative.

Examination of the fundi of five members of the family (all, with the exception of the father) failed to reveal any pathologic process.

Ophthalmologic examination. Right eye. Vision with correction was 1.0. External examination and fundus were negative.

Left eye. External examination of the left eye gave negative results. The lens showed some fine subcortical opacities posteriorly. The disc was not remarkable except as regards the vessels. The inferior temporal artery, a short distance from the disc, was markedly dilated and tortuous as it wended its way to the periphery, where it entered a lobulated reddish-yellow mass. In its central portion the artery became sacculated and tortuous, the sacculations being closely coupled. Accompanying the artery was a similar but more dilated tortuous vein which started in the mass.

The retinal mass was situated well out in the periphery below and slightly temporal (fig. 1 A). It was about 3 disc diameters in size and was elevated about 5 diopters. Its surface was lobulated but smooth and of a reddish-yellow hue. No other masses were seen in the fundus, and the remaining vessels appeared to be normal. Between the disc and the macula the retina showed patches of pale yellow-white exudate slightly suggestive of the exudate of retinitis circinata. Above and below the

(the details of application are included in the previous report). The patient was seen intermittently, and on April 3, 1939, the vision of the left eye was still 0.8. There was no apparent change in the lenticular opacity. The tumor mass was definitely smaller and flatter, being elevated between 3 to 4 diopters, and the mass was of a yellowish color. There was also a definite decrease in the exudate. The blood vessels, however, seemed unaltered. These clinical observations were

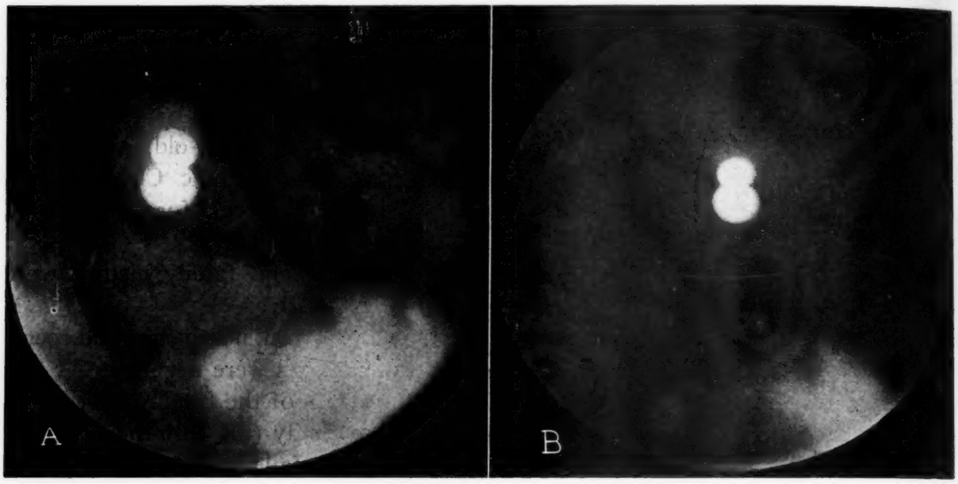

Fig. 1 (Cordes and Dickson). A, angiomatous mass (case 1), in periphery and below, before irradiation. B, mass six months after irradiation.

macula were other small scattered patches of this same exudate. Along the dilated vein were other linear streaks of exudate together with some tiny flecks between the artery and the vein (fig. 2 A).

In the left eye the peripheral fields showed depression above and below for a 3-degree test object at 330 mm. With small test objects (1 degree at 2,000 mm., and 2 degrees at 2,000 mm.) the fields were normal.

The intraocular pressure of the right eye was 19 and of the left eye 18 mm. Hg (Schiötz).

Between September 19th and October 31, 1938, the patient was given 1,202 r

confirmed in colored fundus photographs.

Examination on September 14, 1939, one year after roentgen therapy was started, revealed no change in vision. There was a marked decrease in the exudate in the lower and outer portions of the fundus and between the disc and the macula. The tumor was smaller and much paler than at the previous examination. There was no apparent change in the blood vessels (figs. 1 B and 2 B).

The patient was observed periodically by us until she moved to Seattle. No further changes were noted by us during examinations.

In Seattle, the patient has been under

the observation of Dr. Will Otto Bell, who has kindly kept us informed of his findings. His letter 3½ years after irradiation was started states "the sacculation and tortuosity mentioned in the report have markedly receded. The vein remains somewhat enlarged. The mass measured almost 2 disc diameters in size and roughly shows elevation of from 3 to 4 diopters. No other masses were seen. The patches of pale-yellow exudate above the macula in the original report could not be found, and the linear streaks of exudate above the dilated vein were absent." Vision of the eye was 0.8−3 with correction.

Neurologic examination by Dr. Paul G. Flothow of Seattle was normal except for a positive Romberg.

CASE 2

K. M., a Puerto Rican girl, aged 14 years, was seen at the University of California Ophthalmic Clinic on December 20, 1940. Poor vision had been discovered in her right eye during a routine school examination.

In March of 1937, she had been seen in the Clinic because of "white spots" on the conjunctiva. At that time vision was 1.0−1 correctable to 1.0 in each eye with a small astigmatic correction. Aside from the conjunctiva, the examination was negative. Possible angiomatosis retinae was not suspected, so no detailed search was made in the extreme periphery.

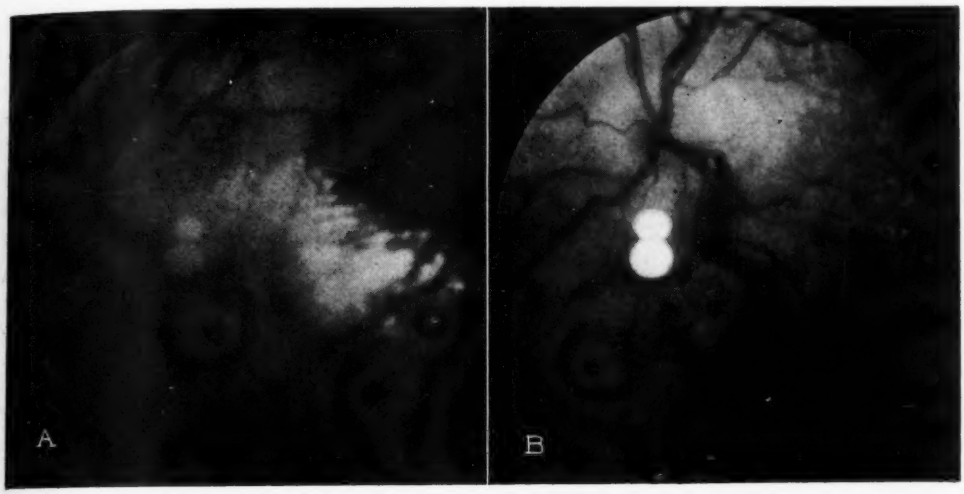

Fig. 2 (Cordes and Dickson). A, area surrounding disc (case 1) and below, before irradiation. B, same area one year after irradiation.

The conjunctival lesion was diagnosed as xerosis of the conjunctiva and responded to vitamin therapy.

The past history was essentially unimportant.

Family history. The father, a Puerto Rican, had died of a brain tumor, three years previously, at the age of 35 years. Examination of his clinic record revealed that on two occasions he had been operated on for decompression and removal of a cerebellar neoplasm, first in 1932 and later in 1934. The pathology department made a diagnosis of angiomatosis cyst of the cerebellum. Recorded examinations of his eyegrounds during his illness revealed changes that were compatible with his in-

creased intracranial pressure. His death occurred in 1937.

Information obtained from the paternal grandmother revealed that the paternal grandfather died "quite young" and that he was blind for some nine months before his death. No further paternal family history was obtainable.

The mother, a Puerto Rican, aged 34 years, was living and well and had no ocular nor cerebral symptoms. As far as could be determined none of her progenitors had any ocular disease or disease of the central nervous system.

Four sisters, aged 4, 11, 13, and 16 years, and two brothers, aged 9 and 12 years, were living and well. Examination of the fundi of all the siblings under homatropine failed to reveal vessel changes or other abnormalities.

Ophthalmologic examination. Vision, R.E., was light perception, unimproved by glasses; L.E., 1.0.

External ocular examination was negative except for an exotropia and poor fixation of the right eye. Slitlamp examination of the anterior segment was likewise negative.

Ophthalmoscopic examination. Right eye. The vitreous was hazy although no distinct opacities could be seen. The disc outline was almost indistinguishable because of edema and grayish-white connective tissue. Leading from the upper part of the disc was a raised gray-white streak that extended to a semisolid-appearing detachment of the retina that was elevated 5 to 6 diopters. The detachment of the retina involved the entire upper periphery and had the appearance as though "it lay over, or was permeated by, coagulated milk." The inferior nasal vessels were dilated and their outlines became lost in a raised spongy-appearing cherry-red nodule about 2 disc diameters from the disc margin. The nodule roughly measured three quarters of a disc in diameter and was elevated 3 to 4 diopters. The vessels emerging from the far side of this mass were much reduced in caliber and quite tortuous. Farther out along their course there were two other smaller, reddish nodules into which both the artery and vein could be seen to enter. Superficial, soft, white exudates obscured macular details (fig. 3).

Left eye. The media were clear. The disc was normal. The vessels were not remarkable except in the inferior temporal quadrant. A branch of the inferior temporal vein was dilated through its entire course to approximately twice the normal diameter. In the extreme periphery this vessel became lost in a reddish-yellow elevated mass about 1 disc diameter in size. No corresponding arterial dilatation was visible. There was one small, sharply defined area of exudate near the inferior nasal vein in the midperiphery (fig. 4).

Intraocular pressure of the right eye was 16.5, and of the left eye 16 mm. Hg (Schiötz).

General physical examination. Physical examination, neurologic, and roentgen examinations failed to reveal any demonstrable pathology. Intravenous urograms were normal.

Diagnosis. The diagnosis was well-advanced angiomatosis retinae in the right eye with an early lesion in the left eye.

Treatment. In view of the apparent success of roentgen therapy in the previously reported case, a similar course of treatment was given to both eyes after consultation with Dr. R. Stone of the Roentgenology Department.

One week following the completion of the therapy there was slight photophobia of both eyes. No conjunctival nor skin reaction appeared at any time.

On April 24, 1941, the vitreous of the right eye appeared to be a little clearer.

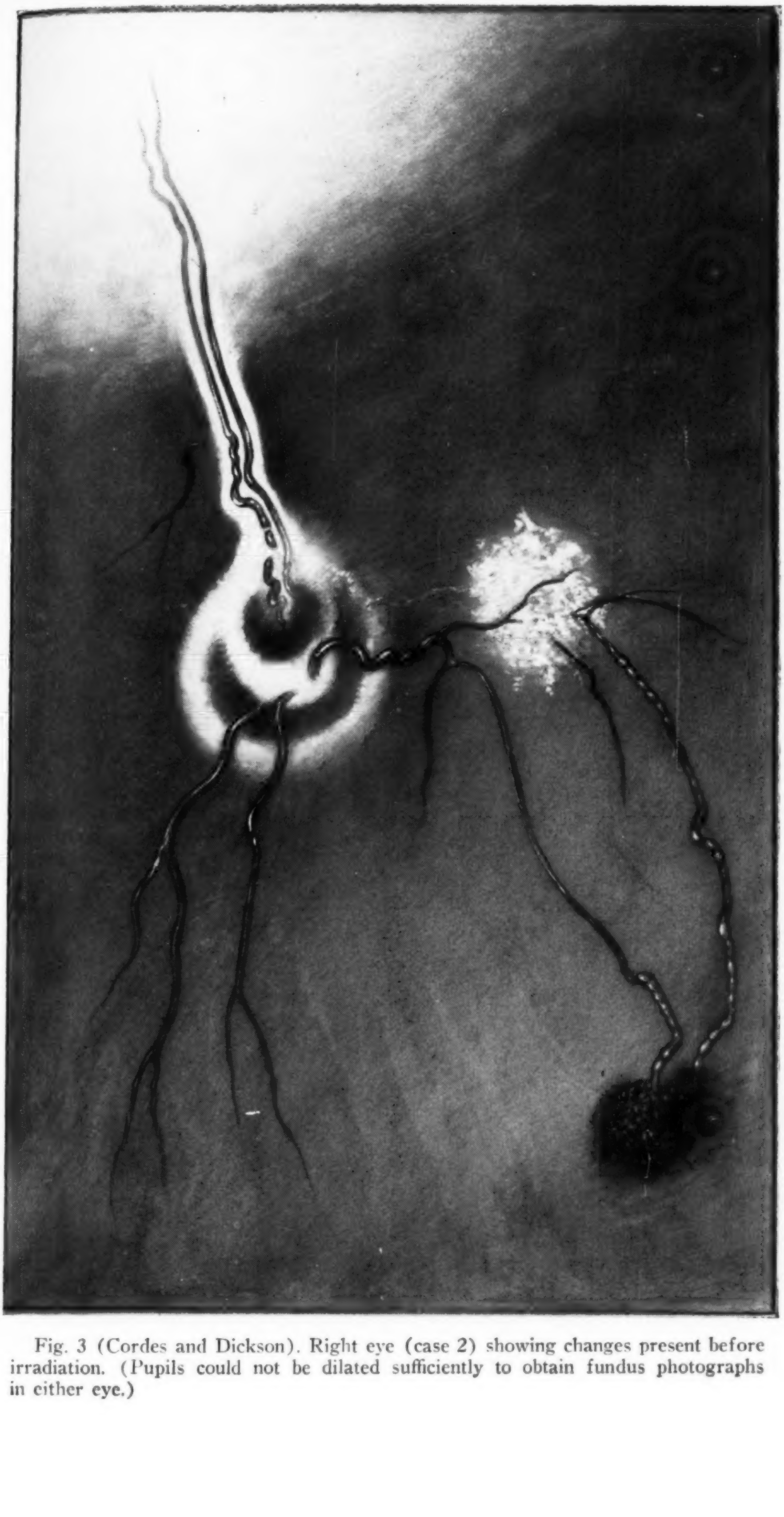

Fig. 3 (Cordes and Dickson). Right eye (case 2) showing changes present before irradiation. (Pupils could not be dilated sufficiently to obtain fundus photographs in either eye.)

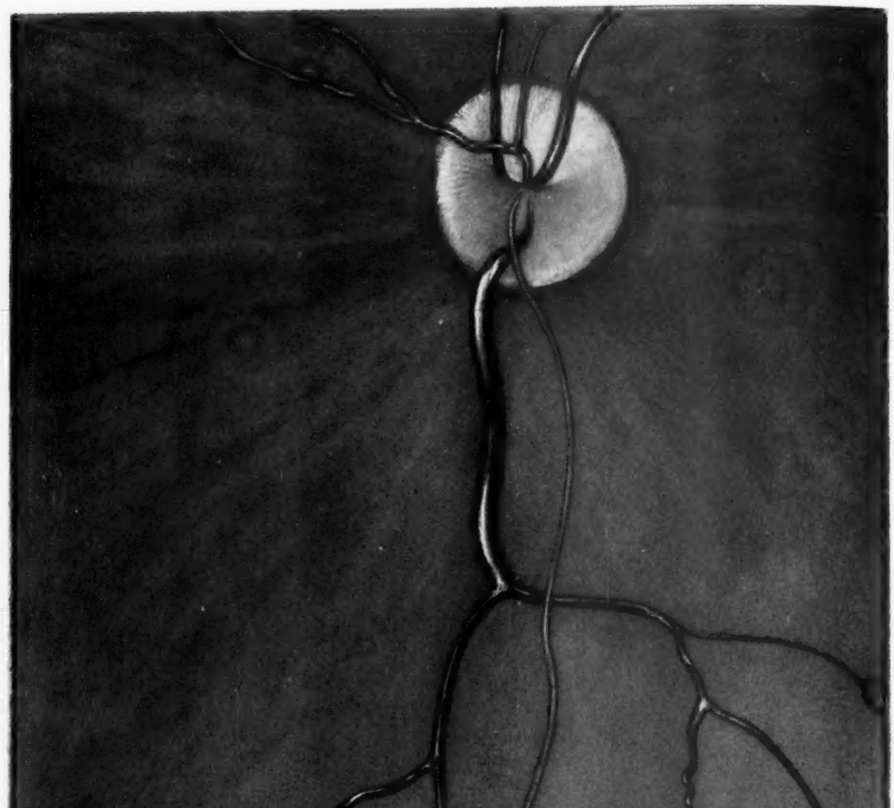

Fig. 4 (Cordes and Dickson). Left eye (case 2), before irradiation.

The disc was still obscured, and the reddish globular mass nasal to the disc appeared flatter, showing only 2 diopters of elevation. The peripheral margins seemed paler. The vessels leaving this area seemed narrower and showed isolated perivascular white plaques along their extent. The other isolated tumor nodules had not changed appreciably. The edema and exudate at the posterior pole persisted. The extensive lesion in the upper periphery showed no change.

The lesion in the left eye appeared to be the same.

Vision, R.E., was light perception; L.E., 1.0.

The ocular tension in each eye was 16.5 mm. Hg (Schiötz). On June 5, 1941, three months after irradiation, the right eye remained unchanged, but in the left eye the nodule in the periphery of the fundus was flatter and whiter in appearance.

On January 30, 1942 (nine months after irradiation), the right eye was amaurotic; the vision of the left eye was 1.0.

The right eye showed some increase in the exudate and in the detachment but otherwise seemed unaltered.

In the left eye the nodule was more circumscribed and whiter, as though scarring was taking place.

On August 24, 1942, the vision of the left eye was 1.0, while the right was, as previously noted, amaurotic.

The picture of the right eye remained unaltered from the previous examination. The lesion in the left eye was smaller, flatter, and more circumscribed; the vessel dilation seemed about the same. The white exudate along the inferior nasal vein has remained unaltered.

TABLE 1

DETAILS OF ROENTGEN THERAPY

Date	Target skin d.	Filters mm.	Kv.	MA	Min.	r air	Total	r Orbit
2-18-41	80	2 Pb	1,000	1.5	2.3	50	50	R-L
2-19	80	2 Pb	1,000	1.5	4.7	100	150	R-L
2-20	80	2 Pb	1,000	1.5	14	300	450	L
2-21	80	2 Pb	1,000	1.5	14	300	450	R
2-24	80	2 Pb	1,000	1.5	14	300	750	L
25	80	2 Pb	1,000	1.5	14	300	750	R
26	80	2 Pb	1,000	1.5	14	300	1,050	L
27	80	2 Pb	1,000	1.5	14	300	1,050	R
28	80	2 Pb	1,000	1.5	14	300	1,350	L
3-1-42	80	2 Pb	1,000	1.5	14	300	1,350	R
3	80	2 Pb	1,000	1.5	14	300	1,650	L
4	80	2 Pb	1,000	1.5	14	300	1,650	R
5	80	2 Pb	1,000	1.5	7	150	1,800	L
5	80	2 Pb	1,000	1.5	7	150	1,800	R

Intraocular pressure, both eyes, was 16.5 mm. Hg (Schiötz).

The patient returned on October 16, 1942, with a small vitreous hemorrhage in the right eye. Aside from this the examination remains unaltered.

The patient was last seen on December 15, 1942, at which time the right eye showed a more extensive detachment with increased gliosis. The intraocular pressure, which had always been approximately 16.5 mm. Hg (Schiötz), had gone up to 30 mm. The condition of the left eye was unaltered from that found on examination on August 24, 1942.

COMMENTS

The therapy of angiomatosis retinae

appears to be limited to electrolysis, diathermy, or irradiation. It is quite apparent that any method employed is feasible only in early cases before detachment of the retina and gliosis have developed.

Electrolysis and puncture diathermy have certain disadvantages in that there is a definite risk of severe hemorrhage, especially if an attempt is made to coagulate the markedly dilated vessels. Judging from some of the photographs the resultant destruction of the retina is extensive.

The advantages of radium over X-ray therapy do not seem to be sufficient to warrant the difficulties attendant to its use.

X-ray irradiation can be given by any competent roentgenologist. It seems safer and less destructive to the retina than do some of the other methods. If its use has not been successful, the other methods may still be employed.

Many men are still reluctant to use irradiation therapy in and about the eye. The tolerance to irradiation increases with the age of the patient. The intensity of the irradiation is greatest in the anterior structures of the eye but becomes proportionately less in its posterior structures. The tolerance of the various structures of the eye has been carefully worked out and is comparatively well understood. The most dreaded complication of irradiation therapy for ocular lesions is lenticular opacity, a condition that can be corrected by operation and therefore should not be considered too seriously if by irradiation the function of vision may be preserved. Clapp[22] could find only 34 cases of postirradiation cataracts up to and including 1932, which includes the period when irradiation of an eye was not so well understood as it is today.

Summary

Three eyes of two patients with angiomatosis retinae were irradiated. In the first patient, who had an early lesion in one eye, there was a marked improvement with retention of 0.8 vision, 3½ years after irradiation. In the second case both eyes were involved, the right eye being in a well-advanced stage, whereas the lesion in the left eye was an early one. Both eyes were given 1,800 r. The early lesion showed definite improvement with vision of 1.0 two years after irradiation. During this time the eye having the more advanced lesion became progressively worse, going on to amaurosis, complete detachment of the retina, and gliosis.

From our experience with three eyes and from what is reported in the literature it appears that in early cases X-ray therapy does offer a convenient, safe means of treating these lesions. The advanced lesions have not responded to any type of therapy.

Electrolysis, diathermy, radium, and X-ray irradiation have all been used with success. It seems worthwhile to record the results obtained so that at a later date it may be possible to evaluate better the various methods now employed in the treatment of this rare disease.

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ORTHOPTICS: EDUCATION IN BINOCULAR SKILL

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No one who understands the normal functioning of binocular vision can fail to realize that it involves a great deal of that kind of muscular coördination we call skill. Like many other normal body functions this skill usually develops unconsciously; it does not need the direction and attention of the individual. Examples of such functioning are the locomotor skills and some of the manipulative skills. The normal individual develops his binocular skills as part of his growth processes and is never conscious of any need to give the behavior of his eyes any special attention.

Not all individuals are so fortunate as to develop normally, however. Binocular vision may never be achieved, or it may be interfered with through disease or accident. Lack of binocular vision is usually manifested by a deviation of one, or rarely both, eyes, from the normal position. Hence the correction of such a condition has been chiefly concerned with straightening the eyes, and the term "orthoptics" adopted for such treatment. It is my contention that this is fundamentally a faulty attitude toward the problem. We are really concerned with the functioning of the eyes, not the position of the eyes. It is true that the eyes cannot function correctly together if they are in an abnormal anatomic relationship, but it is important to realize that even when they are anatomically and mechanically normal they may not function correctly. Achieving a straight position is no guarantee of binocular skill. If, however, a patient has binocular skill he can often control eye deviations.

Skill is neuromuscular coördination directed toward the easy achievement of a specific activity. The individual wants his body to do something, and do it well. Due to individual differences there is wide variation in the skills it is possible for different people to learn. Some people can never learn to be first-class pianists because their hand structure is wrong; some cannot be aviators because their reaction time is too slow. Such examples might be multiplied indefinitely. Even

simple, and apparently essential, bodily functions, like binocular single vision, may be beyond the ability of the patient to achieve. If he does achieve it he is the possessor of a very definite skill.

It is the function of orthoptics to help a patient achieve all the binocular skill of which he is capable. What are the causes of failure?

One of the very serious problems of orthoptics is the present uncertain status of prognosis. What can we do to establish reliable criteria for the selection of cases to train orthoptically? I believe the recognition of the skill function of orthoptic training will help greatly in successfully classifying patients, as well as make training give better results.

Orthoptic training cannot alter anatomic structure, either manifested in the single eye or in binocular relationship. Refractive errors, birth injuries, other traumas, congenital defects, and amblyopias are not subject to correction by exercises or the development of skill. (Amblyopia ex anopsia often comes under the direction of the orthoptic technician, but is preliminary to, rather than a part of, orthoptic training.) This leaves the large group of muscle imbalances, manifested as phorias or tropias, either without surgery or postoperative, and accommodative failures. By the latter group is meant not only hypermetropic squints but patients who show a lack of balance between accommodation and convergence—such as, convergence insufficiencies, exophorias at near, divergence excesses—and adults who find adjustment to bifocal lenses difficult. All these patients show a lack of skill in binocular eye control. When skill is developed the eye position is corrected and the patient is orthoptically cured, as long as the skill is used.

The problem of orthoptic training thus becomes the problem of teaching binocular skill and getting the patient to use it when acquired. The second step by no means follows the first. This has been a common cause of dissatisfaction with orthoptic training. It should be fully recognized that gaining a skill—as demonstrated, for example, by satisfactory orthoptic measurements on instruments—is not proof that a patient is using his eyes correctly on casual seeing. The criterion for successful orthoptic training should be use and not measurement. It is in this field especially that orthoptics deviates from customary medical procedure and enters the field of educational technique.

No teacher, soundly trained in modern educational methods, is satisfied to have his pupil a mere storehouse of information. He wants to make him a useful member of society with behaviors and skills that make him meet his daily problems in a satisfactory manner. If he is to do this he must use what he learns in his own environment, overcoming his own obstacles and not in some ideal and teacher-controlled situation, where all problems are made easy and graded to his ability. A skill which is easy if all the surroundings are favorable is often very hard when the need to use it is thrust suddenly upon the individual under difficult conditions. This factor is part of the definition of skill, under the conception of skill as meaning easy achievement of the required activity. Until it is easy it is not yet really a skill, but only a goal toward which the individual is still working.

The acceptance of this attitude toward orthoptics holds the promise of profound benefits. We need to keep this point of view in mind at all times when working with a patient, to make sure that he does not merely assimilate a bag of tricks to be demonstrated upon request, but is really building up a pattern of eye behavior which is the very peak of his in-

dividual ability and is so easy that he finds it at least as easy as, if not easier than, wrong eye behavior.

This individual ability is very important in orthoptic prognosis and results. Some patients start with handicaps which will prevent their ever achieving perfection; they can learn only partial skill, such as becoming binocular for near but not for distance. Others may be able to practice their skill part of the time but find it too difficult for habitual use. It is necessary to recognize when a patient has achieved all that may be reasonably demanded of him and not keep him working at tedious tasks beyond his ability. Sometimes surgery, or a new refractive correction, can help him further on his path to acquiring binocular skill.

At all times the orthoptist should have a clear appreciation of how the patient is using his eyes in habitual seeing, and of what the next step needs to be.

A great advantage of regarding binocular vision as a skill is the elimination of unnecessary drill in skills the patient already has. For example exercises in eye motility have been frequently prescribed for patients who already have ample eye motility. To practice this particular skill will get them no further on the road to binocular vision, which calls for binocular functioning of a very different kind.

Analyzing the skill status of a patient is different from analyzing his anatomic status, and it is a prerequisite to successful skill training. The examiner must find out what the patient is doing in his casual seeing, and, starting from that point, develop better skills. The lowest form of binocular skill is monocular skill! This paradox is often ignored. Before the patient can be expected to use two eyes skillfully he must be able to control one eye. If he cannot fixate with one eye he cannot develop the more complicated skill of fixating with both eyes. Too often measurements and analyses of a patient's status are made on instruments requiring binocular fixation in order to function as intended, and conclusions are drawn without the patients' ever having looked into the instrument in the manner necessary for valid results.

All instruments on the stereoscope principle are liable to this fundamental error. It has often been taken for granted that if a picture is put in front of each eye, each eye will look, and a report of what the patient sees is a report of how the eyes are behaving as to fusion, depth perception, amount of deviation, and so forth. Unfortunately, binocular behavior is not so simple as this. If the patient has never had, or has lost, the ability to fixate with both eyes at once he does not really experience binocular perception under such conditions as a stereoscope offers, and he has to interpret what he sees in the light of what he recognizes from everyday experience. If he is accustomed to alternate he will alternate during his tests and his responses will sound satisfactory because he has become used to looking in that manner and is adjusted to it. He will report a bird as being in a cage or two lines perpendicular to each other as crossing. This will also be true if his adjustment to his deviation has taken the form of anomalous correspondence (abnormal correspondence; false projection). Before his answers can be accepted as evidence of binocular status it is very important to know what type of adjustment he has made to his binocular problem. For this analysis a close observation of his eye movements under controlled situations is necessary. The observer should find out first whether the patient will look with either eye steadily. I am not concerned here with a nystagmus, but with those random eye movements typical of children who have poor

binocular habits. If presented with an object at which to look they are unable to fixate on it while the observer counts to 10 or even less. The eye jerks away, may return to fixate again, but fails to show control. This condition is much aggravated by inattention in young children, but can be freqently observed apart from any disciplinary situation. If the patient is to learn the skill of binocular vision he must first learn to control his eyes under the easiest and simplest conditions. The sequence is one eye, either eye, both eyes. To plunge him into the highly skilled task of beginning with both ,eyes is to court failure. This does not mean that he is to be kept at the task of using only one eye until that skill is perfected. We do not learn perfect manual skill with one hand, as on the piano, or typewriter, before starting to train the other hand. As early as possible in his training the patient should experience the satisfaction of using both eyes. Teaching skill consists in presenting tasks which are not beyond the ability of the patient, but which encourage him to try his best to fulfil; in making him try to do a little better than he has ever done before. If faced with a situation he cannot meet, he tends to make no effort at all, or to "fake" his responses in order to gain approval.

To stimulate and encourage looking with two eyes it must be made easy for the patient, and this is where a major amblyoscope is invaluable in orthoptic training. The targets can be readily adjusted to fit his eyes; it is not necessary for him to adjust to the machine. In this way he gets the experience of binocular vision under the easiest and most favorable conditions. It is failure to reach this first stage which frequently causes orthoptics to be condemned as unsuccessful. *No amount of work on orthoptic equipment is of any value to the patient unless both eyes are fixating on the maculae.* This is the absolutely essential first step in successful orthoptic training.

It is at this first stage of training that correction of anomalous correspondence occurs. If the patient does not fuse when fixating steadily with the maculae, but recognizes fusion as existing when one eye fixates eccentrically, then such anomalous correspondence must be corrected before further binocular training is undertaken. A large amount of stimulation by tested techniques has proved successful in correcting this condition.

The patient is now ready for the development of amplitude. This is not merely a skill demonstrated on instruments or prisms, but is the absolutely necessary means by which the patient uses binocular vision for comfortable every-day vision. Without amplitude of fusion he cannot adjust to the problems of casual seeing, which require rapid adjustment of binocular fixation, with its consequent change of the fusion angle.

The patient who has always depended upon monocular fixation has no idea of what it feels like to "hang on" with both eyes during a change of angle. He usually cannot recognize whether he is doing so or not. Suppression of one eye sets in so instantly and smoothly that the change from binocular to monocular fixation occurs quite unconsciously. That is why orthoptic training usually takes a long time. A great deal of practice has to be given to the recognition of diplopia. Before the patient can transfer his binocular skill from orthoptic instruments to every-day habits he needs to recognize his success or failure at binocular fixation, and his early experience, before his eyes are straight, will result in diplopia when he is successful. When he gets diplopia on casual seeing he can help educate himself. He is in the position of a musician who hears a discord, or a typist

who sees the wrong letters appear on her page; there is something uncomfortable in his experience and he has an impetus to correct it. Now he *must* either learn to straighten his eyes (fuse), whether by more training, or surgery, or else relapse into suppression.

The balance of his orthoptic training should give the patient enough practice in using amplitude—convergence and divergence—to make it easy for him. This is the point at which phoria patients often show their failure. They may be able to demonstrate good amplitude when they try, but it is not easy for them. Orthoptics can give them greater comfort by teaching them more skill; that is, greater ease of amplitude. It is not the amount of amplitude alone that decides the visual comfort and habits of a patient, but the ease with which he does it; the perfection of his neuromuscular coördination.

Conclusion

Orthoptics is not exercising of the ocular muscles; it is not even primarily a procedure designed to straighten the eyes. It is the teaching of a patient to use his two eyes together for comfortable binocular vision. The emphasis in such training is the teaching of a skill in the use of unskilled neuromuscular coördination. Orthoptics will be of much greater value to physicians and their patients when its function is re-evaluated: not as primarily concerned with anomalies of eye position, but as devoted to helping the patient learn comfortable binocular visual habits.

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LENS EXTRACTION FOLLOWING FILTERING OPERATIONS FOR GLAUCOMA*

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Every ophthalmologist of experience is confronted occasionally with the problem of senile cataract developing slowly or progressing after a successful operation for glaucoma. When the previous operation has been iridectomy or cyclodialysis the problem is not so serious, since the lens may be extracted by one's customary method without much danger that increased intraocular pressure will recur. When, however, a filtering operation has been performed, the difficulties are obvious. Due to these difficulties, operation is usually postponed as long as possible, and one sees patients who have been severely disabled for years before a decision to resort to surgery is made.

Some ophthalmologists have performed extraction above, cutting through or beneath the bleb, and some have reported no return of increased tension after this procedure. Others have extracted the lens from below, without disturbing the bleb. If a complete iridectomy has been performed previously, and especially an iridencleisis, this procedure becomes difficult or even impossible. One must either tumble the lens over the pupillary border, which may be rigid or even fixed to the lens capsule, or else perform a second iridectomy below, dividing the iris into two halves with complete destruction of its sphincter function. In the practice of the late Dr. Harold Gifford, I remember having seen this procedure employed in several cases and always, as I remember it, with complications resulting from incomplete removal of cortex or, in at least one case, loss of vitreous due to difficulty in getting the lens to present.

To avoid these difficulties McLean[1] has recently described a method of extraction from above which avoids disturbance of the bleb. The incision is made in clear cornea well in front of the bleb after the insertion of a suture on each side, by McLean's method, which employs a groove cut half-way through the cornea. This insures accurate closure of the wound, and was successful in his hands in four cases, three following trephining and one following iridencleisis. He states that other surgeons at the Wilmer Institute have followed this procedure with equally good results, without disturbance of the filtering scar. In such cases, intracapsular extraction is practiced whenever possible. Aside from the technical difficulty of making the section come out exactly in both grooves without cutting either suture, which should not be serious for a skilled operator, the necessity of making the section so far forward as not to disturb the bleb would seem to present a serious difficulty in certain cases. It cannot be easy to extract the lens in its capsule over such a shelf of cornea as must be left, should the bleb involve a considerable portion of cornea. I have tried a somewhat similar procedure, as described by Tieri,[2] in which the central portion of the section is made in front of the bleb with the keratome, and enlarged at both ends by scissors under a specially prepared pocket flap of conjunctiva. In spite of care not to disturb the bleb, either the proximity of the corneal section to the bleb or the disturbance of neighboring conjunctiva was responsible for complete loss of the bleb in the two cases in which this procedure was employed.

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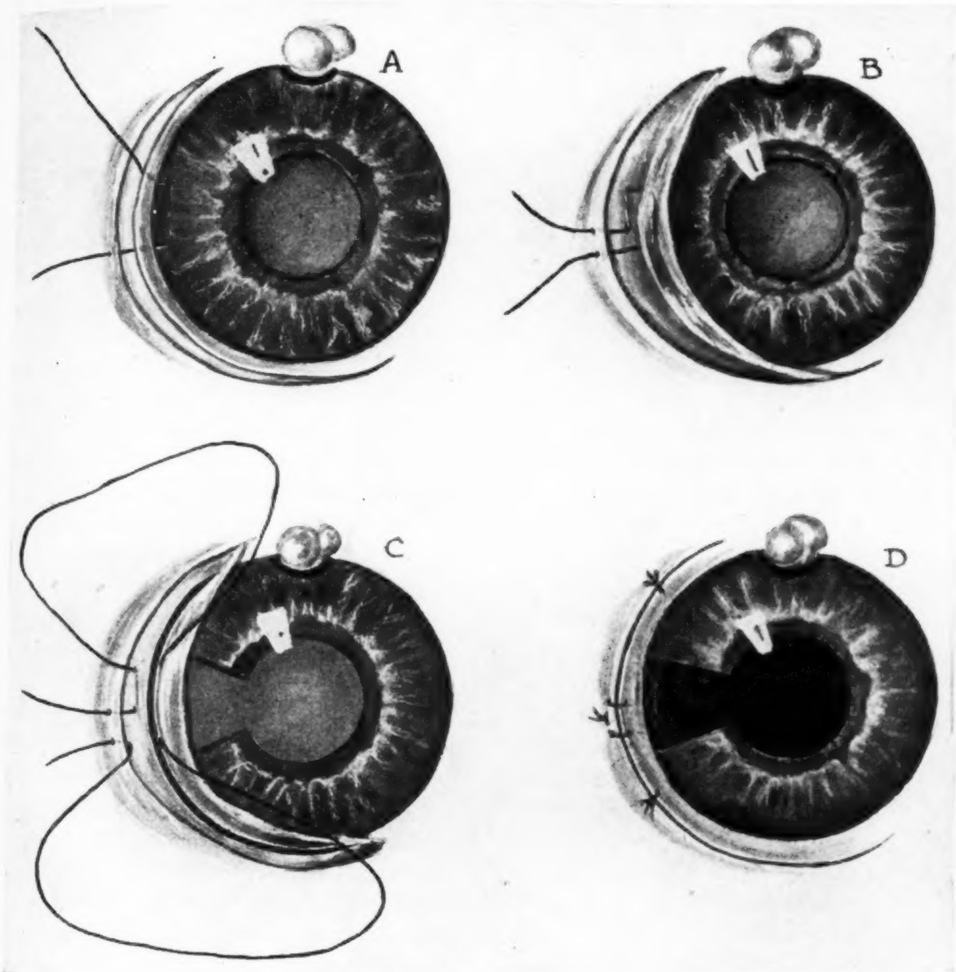

Fig. 1 (Gifford). Technique of incision and sutures employed in extraction of lens from the temporal side.

Van der Straeten has described certain advantages of a section at the temporal side, which he seems to have preferred for ordinary cataract extractions. He did not employ it, however, after filtering operations, preferring a section in the cornea in front of the bleb.[3] The temporal area, he pointed out, is easily accessible, and a section in this location is splinted by the lids, so that it does not tend to gape. While not convinced of the advantages of a temporal section in the usual cataract extraction, I was impressed by its possibilities under the special conditions discussed in this paper, and have employed it in seven such cases to date, and in an eighth case of a different type, with results which will be described. In all but one of the cases the cataract was of the normal senile variety, developing over a period of years following operation. In four cases it was known to be present before the filtering operation was performed.

Since it is desirable to avoid entirely the vicinity of the bleb and since a large

enough section for intracapsular extraction is desirable, the section is not made exactly at the temporal side but occupies the section from the 11- to 5-o'clock position on the right eye and the 1 to 7 on the left eye. A corneoscleral suture is as desirable as in the usual extraction, and this has been employed with slight variations in its relation to the conjunctival flap. What has seemed to be the most practical method is shown in figure 1. The conjunctival flap, prepared with scissors, is small, and the corneoscleral suture is reinforced by two conjunctival sutures above and below. Fixation is on the superior and external recti muscles, the former being held by fixation forceps and the latter by a suture. The section is made with the usual knife from below, but on account of the unusual location it is not so easy to make a large section as when this is made above. Hence it is usually made fairly small and completed with very sharp Stevens scissors. There is no reason why it cannot be made with the keratome and enlarged in the same way by an operator who finds the location awkward or in a patient with a prominent zygomatic arch, and this was done in two cases. Canthotomy is often convenient. When a peripheral iridectomy has been performed, a new complete iridectomy is made at the temporal side. When a complete iridectomy has been made above in trephining, or an iridencleisis has been performed with inclusion of one iris pillar in the scar, the temporal side of the coloboma is grasped with iris forceps and the iridectomy is enlarged to the temporal side. A moderate enlargement in this direction will allow a grasp of the equator of the lens with Verhoeff's forceps, and with the application of pressure at the nasal limbus by means of the ring expressor it can be brought out with the temporal border first, when other circumstances are favorable.

Case reports

A summary of the cases in which this method of operation was used follows.

Case 1. Rev. R., aged 65 years, had undergone bilateral trephining elsewhere a year before. Prior to operation, according to the surgeon's report, vision was R.E. 3/10 and L.E. 1/200. The visual fields were contracted to within 5 degrees of the fixation point in each eye. After operation the anterior chamber of the right eye remained empty for six weeks and the left never re-formed. Cataracts developed rapidly, and vision was reduced to light perception in each eye, with poor projection in the left eye. In the right eye a good bleb was present, and tension was 11.5 mm. Hg (Schiötz). Operation was performed on the right eye on April 25, 1940. It was necessary to free the iris from the lens capsule all around, and in doing so the capsule was injured. Capsulotomy and temporal iridectomy were performed, the nucleus was expressed, and most of the cortex was removed by irrigation. Needling of the secondary membrane was performed three weeks later. Vision a few weeks later was 20/20 with correction, although the field was very small. The tension has remained between 25 and 28 mm. Hg since operation. The surprisingly good central vision suggests that some lens opacities must have been present before the trephining, although none were noted by the previous surgeon.

Case 2. Mrs. E., aged 58 years, had had iridencleisis performed, in 1931, for chronic simple glaucoma of the right eye. The other eye had been operated upon elsewhere, with good result as to tension, but at a time when almost no vision re-

mained. Vision in the right eye remained 20/50, the acuity present before operation, for five years, with tension from 11 to 15 mm. Hg. (Schiötz). Nuclear cataract had begun before this time, and by 1940 vision was reduced to 20/200. Operation for the cataract was performed on October 14, 1940—an intracapsular extraction by the temporal route. A year later vision with correction was 20/60, and tension remained 10 mm. Hg (Schiötz). The bleb was a little flatter than before the extraction but was still functioning.

Case 3. Miss N., aged 66 years, had had iridencleisis performed on the left eye in 1934. The visual field before operation was good, and tension was 32.5 mm. Hg (Schiötz). Opacities in the posterior cortex were noted before operation. Vision after operation was 20/40, and tension was 5 mm. Hg (Schiötz). By July, 1941, the nuclear and posterior cortical opacities had progressed until vision was reduced to 20/100 with difficulty. Lens extraction by the temporal route was performed on July 25, 1941. The capsule tore easily, and extracapsular expression was performed through the enlarged iridectomy opening. No discission was required. At last report, 18 months after operation, vision was 20/20, and tension 24 mm. Hg (Schiötz). The bleb remained the same as before operation.*

Case 4. Miss M., aged 76 years, had had bilateral iridencleisis performed in 1934. Some nuclear lens opacities were noted before operation. Ocular tension averaged 30 mm. Hg (Schiötz) in the right eye, and 38 mm. in the left. Vision was reduced to the ability to detect hand movements in the left eye, but was 20/30 in the right. It remained the same, after operation, for two years, tension averaging 18 mm. Hg in the right eye, and 13 mm. in the left. By May, 1940, lens opacities had reduced vision to 20/300 in the right eye, and extraction by the temporal route was performed through an enlarged iridectomy opening. Intracapsular extraction with the Verhoeff forceps was uneventful. Recovery was complicated by a senile psychosis, but vision after eight months was 20/30. Tension was 19.5 to 22 mm., and the bleb, while smaller than before operation, was still functioning.

Case 5. Mr. F., aged 59 years, had had iridencleisis performed on the left eye, on March 26, 1936. Tension before operation was 47.5 to 55 mm. Hg. (Schiötz), and vision was 20/40 −2. Nuclear and posterior cortical lens opacities prevented a good view of the fundus. Following operation tension was at first 30 to 32 mm., but miotics and massage brought it down to a range of 17 to 25 mm. Vision was 20/40 following operation, but the lens opacities slowly developed, and by 1940 vision was reduced to 20/200. On June 24, 1940, the lens of the left eye was extracted from the temporal side after the temporal pillar of the iris opening had been excised. It was difficult to dislocate the lens, so capsulotomy was performed. After needling, on September 5, 1940, vision was 20/15 with correction, and tension has remained 11 to 17 mm. Hg to date, without the use of miotics. In this case it was necessary to cut the iris on the side of the pillar which was included at the time of iridencleisis. The bleb, which was never large, remained about the same after the secondary operations. The lens of the right eye, in this case, had been opaque following an iridectomy performed by an-

* Report by Dr. Folsom of Fond du Lac, Wisconsin, on December 3, 1942.

other surgeon during an acute attack of glaucoma, and the pupil was drawn up nearly to the limbus. Iridotomy and later extraction of the shrunken lens resulted in vision of 20/15, the tension remaining a low normal.

Case 6. Mr. B., aged 73 years, was referred by Dr. C. A. Thigpen of Montgomery, Alabama, who had trephined both eyes, 12 years previously, for chronic simple glaucoma. Vision in the right eye before operation was much reduced, but in the left it was about 20/30 and had remained the same until recently. When the patient was first seen, in 1939, tension in the right eye was 18 mm. Hg, left 10.5 mm. (Schiötz), and vision in the left eye could be improved to 20/40. Three years later vision was reduced to 20/100 by nuclear and posterior cortical cataract, tension remaining 8.5 mm. Hg (Schiötz). There was a large bleb extending well into the cornea. On April 29, 1942, extraction from the temporal side was performed, incision being made with the keratome and enlarged with scissors. A temporal iridectomy was performed. The capsule was apparently injured by the keratome, so a large capsulotomy was performed, followed by extracapsular expression. Needling of a thin membrane was done on June 16, 1942, and vision, on August 7, 1942, was 20/25 with correction. Tension varied from 8 to 13 mm. after operation, and the large bleb remained unchanged.*

Case 7. Mrs. B., aged 63 years, had iridencleisis performed on the right eye, in 1932. Before operation, vision was 20/20, and tension 32 to 35 mm. Hg (Schiötz). No lens opacities were noted. Tension was reduced by the operation to 12.5 mm. and remained between 12 and 17 mm. A year later, a few lens opacities were seen, but vision remained 20/20 until 1936, after which it failed very slowly, reaching 20/70 in 1942. Extraction from the temporal side was performed November 18, 1942, after an excision of the temporal pillar of the iris. Intracapsular extraction was successful but required more pressure than usual. Vision with correction, after four weeks, was 20/30 and intraocular pressure was 13 mm. Hg. (Schiötz).†

An eighth case may be mentioned, as this method of extraction was employed, although no filtering operation had been performed. The patient was a woman of 74 years, who had lost all sight in the left eye following an acute attack of glaucoma some years before. The right eye showed no increase in tension, but was smaller than normal and had a congenital coloboma of the iris in the typical downward direction. Vision was reduced to 20/200 by posterior cortical and nuclear lens opacities. At operation an incision below and temporally was made, extending from the 10- to the 4-o'clock position; that is, somewhat lower than in the cases previously described. The congenital coloboma was enlarged slightly to the temporal side, and intracapsular extraction was performed without difficulty. The chamber was slow in re-forming but attained normal depth in two weeks, and vision of 20/25 was obtained with correction. Tension was 8.5 mm. Hg (Schiötz) at the last examination, three months after operation.

Discussion

Experience with this group of cases has shown extraction by the temporal route to be a practical method of dealing with the problem of cataract following a successful filtering operation. In only one case (case 3) did tension rise appreciably above the level present before cataract extraction, and the filtering scars showed

* A later note by Dr. Thigpen indicated some reduction in vision but no return of tension.

† Four months later vision is 20/25, and tension 12 mm. Hg (Schiötz).

very little if any change.*

Visual results were satisfactory, the vision obtained being in all but one intance (case 2) as good as at the time of the filtering operation, and in several instances (cases 1, 5, and 6) considerably better. It is evident that such visual results cannot be attributed entirely to this method. Provided the lens is removed without complications, the resulting vision will depend chiefly on the amount of damage done to the eye before the filtering operation was performed. It was simply fortunate that in most cases of this series operation was performed at a relatively early stage in the course of glaucoma.

There were no operative complications directly referable to the method. Vitreous loss did not occur nor was there delayed re-formation of the anterior chamber, hyphema, or iris prolapse in any glaucoma case. In case 8 there was delayed re-formation of the anterior chamber.

In only four of the eight cases was intracapsular extraction possible, and for this the temporal route may be in part responsible. This was certainly not true in case 1, in which the iris was so densely bound to the capsule that it was impossible to free it without injuring the capsule. In case 6, the use of the keratome may have been responsible for injuring the capsule, while in case 3 the capsule was exceedingly friable and would have been torn by any method. Undoubtedly, a cause of failure in certain cases is failure to make a large enough incision when operating by this unaccustomed route. The incision should be enlarged to a full half-section, sparing the region of the bleb and utilizing the region past the 6-o'clock position as far as necessary. It must be admitted that the manoeuver of intracapsular delivery from the temporal side seems more difficult than in the usual method, especially after iridencleisis, when the iridectomy is chiefly above the center of the incision.

It happened that, in the series, five eyes had been subjected to iridencleisis and only two to trephining. Three of the eyes after iridencleisis (cases 2, 4, and 7) were right eyes, in which the nasal pillar was included and the temporal pillar free. In all of these intracapsular extraction was successful. Two (cases 3 and 5) were left eyes in which the temporal pillar was included. This necessitated cutting the included pillar in making the iridectomy. It was feared that this might cause interference with the filtering scar, but this did not occur in either case. In neither case, however, was intracapsular extraction possible, and the impression was given that the iris on the included side was perhaps more rigid and offered more obstruction to delivery of the lens. It seems worth while, in the future, to include the nasal pillar in operating upon either eye, especially when cataract is already present at the time of iridencleisis, leaving the temporal pillar free for possible later cataract extraction. The method of cutting the sphincter on both sides and including a middle tongue of iris would, of course, meet this situation, but is, I believe, open to other objections.

The rather poor average of intracapsular extractions did not affect the final outcome in any of these cases, as the needlings required in three cases were not followed by return of increased tension. It is believed that further experience with the method will improve the average of intracapsular extractions.

* Tension 24 mm. Hg (Schiötz).

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ASPECTS OF BRAZILIAN OPHTHALMOLOGY

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Apparently Wilhelm Pies in the seventeenth century was the first to write extensively about eye diseases in Brazil. Pies was a Dutchman who had been sent for by the ruler of the Dutch settlements in what is now the northeastern state of Pernambuco, on the "Brazilian shoulder," to make a survey of the diseases then prevalent in the colony. Possibly others before him, and surely many after him, have added to the world's knowledge of ocular pathology with their respective contributions to the eye conditions found in Brazil. Some were doctors of medicine, others scientists in the broad sense of the word, and still others were just globe trotters whose reports, therefore, varied in accuracy according to their ability.

As to the practice of ophthalmology: Until the nineteenth century, it was obviously limited to a very few minor eye operations and treatments, all of them performed, of course, by the general practitioner. No medical schools were available in the country before the beginning of the last century, and consequently all the doctors were imported from Portugal and elsewhere.

According to tradition, the first home-trained eye surgeon in Brazil was Alvares Machado, who had his training in a military medical school then existing in São Paulo, and who, after his graduation, in 1819, was appointed military surgeon. Alvares Machado was a man of great ability and quite versatile, proof of which were his successful cataract extractions with Daviel's technique and his no less successful rise in politics, enabling him to become a "Grandee" of the Brazilian Empire and a president of one of its provinces.

In the early forties, ophthalmology was still practiced sporadically, as a side line of general medicine and surgery, but this did not prevent Martins in Rio from being affected by the mania of the time for operating on the cross-eyed by the method of Dieffenbach, and reporting in 1841 that he had performed 82 tenotomies.

Carron du Villards, a French ophthalmologist of Italian extraction, who had studied with Scarpa, came to Rio in 1856, and remained there until his death in 1860. He was responsible for stimulating an increased interest in ophthalmology among Brazilian physicians. He had been a great traveller, at first in Europe, where he visited both the Scandinavian and the Balkan countries, then in Africa, in Tripolitania, Sierra Leone, and Liberia; later in the Western hemisphere, where he visited Cuba, establishing himself there for two years, and in Mexico, where he became the head of the health department and had the rank of a general. Carron du Villards stimulated the progress of ophthalmology rather through his enthusiasm in imparting his knowledge to others than by his own personal achievements. In Rio he published a pamphlet in Portuguese, entitled "Adversaria ophthalmologica" and "Passe temps ophtalmologiques" in French, both in 1859.

Carron du Villards, an accomplished ophthalmologist, familiar with Helmholtz's new technique of using a mirror to see the fundus, a past master in cataract operations (he had operated on more than 4,000 patients during his stay in Cuba, Mexico, and other Latin American countries), fostered great enthusiasm among Brazillian would-be ophthalmologists. Gama Lobo was one of the first to emulate the Savoyian oculist; after studying in Germany with Arlt, on his return to Rio he was put in charge of the first out-

patient eye department established in 1863, at the charity hospital (Santa Casa).

Gama Lobo showed great interest in ophthalmology, and at the IV International Congress in London read a paper on his measurements of the distance between the cornea and the posterior surface of the lens, to accomplish which he had, acting on Helmholtz's advice, used sun rays passing through a slit in a dark room.

Hilario de Gouvea was another of the early Brazilian ophthalmologists. Gouvea in 1867 studied with A. von Graefe and later was assistant at Becker's eye clinic. Returning to Rio in 1873 he practiced and taught ophthalmology. His teaching was unofficial, though, for no chair of ophthalmology was established in the medical school of Rio until 1881, but Gouvea had the encouragement of the dean of the medical college to impart his knowledge of ophthalmology to the medical students long before the chair was finally established, and he became the first full professor of ophthalmology in Brazil.

Correa Bittencourt, another of the early ophthalmologists, wrote a book on the relations of general diseases to the eye, one of the three works ever written on the subject up to that date. Moura Brasil, one of his contemporaries, claimed priority in describing the use of the Abrus precatorius against pannus in trachoma, de Wecker making the same claim. Many other doctors became interested in ophthalmology in the early eighties, and this growing interest could be sensed by an ambitious project to start an ophthalmologic journal, which materialized, in 1888, as the "Revista Brasileira de Oftalmologia." No special medical society existed then in Brazil, but at gatherings of general medical societies there were several meetings for the exclusive discussion of ophthalmologic matters. The first efforts in a prevention of blindness campaign were contemporary with these early developments of Brazilian ophthalmology, and in the late eighties, in several papers, theses, and lectures (Ottoni, G. Alvaro, and others), the universal use of Credé's method for the prevention of ophthalmia neonatorum was advocated.

In the first three decades of the twentieth century interest in ophthalmology grew steadily but in a less spectacular way. In the Medical School in Rio, Abreu Fialho was head of the department of ophthalmology, and a very large number of younger Brazilian ophthalmologists became Fialho's alumni. At the Medical School of Bahia, the oldest in Brazil, S. Pereira held the chair of ophthalmology, but to Cesario de Andrade, who succeeded him, goes the credit of training the great majority of ophthalmologists who now practice in the northern part of Brazil. In those days, however, it was customary for the young and ambitious ophthalmologists to have their "grand tour" before establishing themselves in private practice. Their preference was divided between France and Central Europe (Germany, Switzerland, and Austria); at first France seemed to be most popular, but gradually more and more Brazilian specialists, overcoming linguistic difficulties, had their training in Central Europe.

In 1918, at a special medical gathering in Rio called to discuss trachoma problems, an ophthalmologic society, the "Sociedade Brasileira de Oftalmologia" was founded. This society, however, did not acquire national status, and its meetings were not very frequent until the late twenties, when greater enthusiasm prevailed and an ophthalmologic journal, the "Annaes de oculistica do Rio de Janeiro," made its first appearance. Ophthalmologic journals in Brazil until then had not come to stay and the "Revista Brasileira de Oftalmologia" had long since discontinued its publication. In this respect it is rather appropriate here to quote Axenfeld,

who was always sarcastic in his remarks on the appearance of new ophthalmologic journals in Europe, which he considered to be more an expression of personal vanity than of the needs of the specialty. There was, however, one exception he liked to emphasize, and that was the Western hemisphere, where the appearance of new journals seemed justified to him.*

With the fourth decade of this century the progress of ophthalmology in Brazil gained a new impetus. An ophthalmologic society was established in São Paulo in 1930, and in several other large cities such as Porto Alegre, Bello Horizonte, Bahia, and Campinas ophthalmologists started their own societies, in some cases jointly with otolaryngologists as the habit of practicing the two specialties simultaneously prevails in the smaller towns in Brazil as elsewhere on this Continent. In 1935 the Ophthalmological Society of São Paulo called a gathering of Brazilian ophthalmologists, which proved to be a great success, and it was decided that such meetings should be held regularly every two years, and thus the second Brazilian Congress of Ophthalmology met in 1937, in Porto Alegre, the southernmost state capital, the third met at Bello Horizonte, the capital of an inland central state, in 1939, the fourth in Rio, in 1941, and the fifth is scheduled for this year in Bahia, a northeastern state.

At these national congresses ophthalmologists from every part of Brazil convene, and, following the system adopted in International Congresses of Ophthalmology, two or three main subjects are chosen beforehand, and a few prominent ophthalmologists are selected to report on them. But besides these so-called "official" papers there are sometimes as many as 150 or more papers on different subjects. The quality of the papers varies considerably, but the leaders of Brazilian ophthalmology feel that it is better to have a crowded program and encourage ophthalmologists to write papers and come to the meetings, than to have an excellent program but to have to exclude papers whose authors, living in secluded communities, would benefit from attending the congresses.

From the very first the Brazilian national ophthalmologic meetings had an international leaning, and at the first congress in São Paulo papers sent in from the United States, Germany, and France were read *in absentia,* while Argentinian colleagues came all the way from Buenos Aires to participate. At the meetings which followed the attendance of colleagues from other countries increased, and it should be remembered, too, that it was at the meeting at Bello Horizonte in July, 1939, that the first authorized suggestion for a Pan-American gathering of ophthalmologists was made; this suggestion materialized the next year in Cleveland, thanks to the sponsorship of the American Academy of Ophthalmology and Otolaryngology, which undertook the organization and accepted the financial responsibility for the meeting.

The proportion of clinical reports, reviews of given subjects, research papers, and papers on social aspects of ophthalmology, such as prevention of blindness, compensation, and others, has varied in the several meetings of these Brazilian Congresses of Ophthalmology. Generally speaking, it may be stated that, fortunately, the papers reporting clinical cases are on the decrease in favor of reviews,

* The following journals of ophthalmology are published in Brazil: "Revista de Oftalmologia de S. Paulo," "Arquivos Brasileiros de Oftalmologia," "Oftalmos," "Revista Brasileira de Oftalmologia," and Arquivos Brasileiros de Profilaxia da Cegueira." The "Arquivos do Instituto Penido Burnier" deals with other subjects also.

research papers, and papers on prevention of blindness, ophthalmologic hygiene, compensation, and the like.

At the meeting of the IV Congress in Rio, it was decided to establish a Board of Ophthalmology which would, in a way, be the amalgamation of the idea of the American Board of Ophthalmology and the British Council of Ophthalmology. The Board has now started its work, and it is hoped that the fine results achieved by the Board in America will be duplicated in Brazil. Provision has been made in the bylaws of the Brazilian Board of Ophthalmology for increased facilities for the training of doctors who wish to become ophthalmologists. So far, such facilities are not adequate. Those who have decided to practice ophthalmology start their training by working in the eye ward or in the eye out-patient department, first as voluntary assistants, and then possibly as assistants. In the latter case their training may be very good, but if their stay is not sufficiently long the knowledge they acquire may not reach the desired minimum. Indeed in only a few departments of ophthalmology is there provision for scheduled routine training, and in general new ophthalmologists get their training as best they can according to their own judgment. Postgraduate teaching is making headway, however, and regular courses and training possibilities are improving.

Regarding the prevalence of eye diseases in Brazil, it is hard to generalize. For one thing, there are not as yet reliable statistics about the incidence of the several ocular ailments in all the hospitals and out-patient departments throughout the country; and, for another, in many less-developed sections of the country there are no facilities for the examination of patients at all.

In spite of this there is a general feeling, based on existing data, that some conditions are rarer or more common as the case may be in Brazil than in other countries. Thus retinal detachment and uncompensated glaucoma seem to be less common, whereas the incidence of pterygium appears to be greater (about 5 percent of all eye patients) than elsewhere. Regarding refraction, available statistics among eye patients of dispensaries and eye out-patient departments, or among school children, show that hypermetropia is about five times as common as myopia in children and about three-and-a-half times as common in all patients taken indiscriminately.

Acute catarrhal conjunctivitis is generally caused by the Koch-Weeks bacillus and not so frequently by the Pneumococcus.

As regards eye diseases that are found in Brazil as in other parts of the world a few remarks can be made about certain facts which show variations in some of these diseases. Thus, epiphora due to abolished drainage of tears, as a consequence of extirpation of the lacrimal sac, is not a frequent complaint probably because the absence of low temperatures does not cause increased lacrimation and consequent epiphora. Trachoma is seldom complicated with purulent conjunctivitis as in Egypt or Palestine. It appears to be the cause, due to its complications, of about 10 to 12 percent of the cases of blindness in the sections of the country where it is endemic (a section in the northeastern states where it has been found since the eighteenth century, and in the southern states where it was imported with immigrants from the Mediterranean countries and Japan). "Ophthalmia Brasiliensis," a disease with corneal involvement and hemeralopia caused by lack of vitamin A and often described by ophthalmologists in Brazil in the last decades of the nineteenth century, is now quite rare.

Uveitis in Brazil as elsewhere is

caused chiefly by syphilis, tuberculosis, or focal infection. Until two decades ago lues was generally regarded as the cause in the great majority of cases and therapy was regulated accordingly. Lately attention has been increasingly called to other possible etiologies, such as foci and tuberculosis. This latter etiology is accepted by the patient with difficulty, as he shuns the idea of being affected by tuberculosis but minds less being told he is syphilitic.

Indirect ophthalmoscopy, using an external source of light, is still very popular, and in many instances is the routine method of examining the fundi. The electric ophthalmoscope, however, is finding its way, and the younger men in the more progressive schools of medicine, even those who are not going to practice ophthalmology, are being trained to use this instrument. The measurement of refractive errors to a great extent is managed in very much the same way as it is in continental Europe. The use of cycloplegics for that purpose is not so universal as it is in the United States, but the trend is toward more accurate measurements, and more and more ophthalmologists are using cycloplegics as a matter of routine every year. As in continental Europe, less attention is paid to muscular imbalance, and fewer efforts are made to correct this condition than in the United States. As a sign of the prevailing interest in the study of refraction, there is the fact that a recent textbook on this subject, edited by a young Brazilian ophthalmologist, has met with considerable success.

According to the law and to prevailing ethics there should be no connection whatsoever between the ophthalmologist who prescribes and the optician who dispenses the glasses. Until very recently opticians attended no regular courses on the subject, merely learned their trade as apprentices.

Regarding strabismus, the approach of the rank and file ophthalmologist to the subject is more or less the same as that prevailing in continental Europe. Until recently, indeed, very few efforts were made to achieve physiologic cures, and operative treatment performed after adolescence was generally for cosmetic purposes. There now seems to be, however, some interest in changing this approach to the subject, proof of which is the fact that strabismus was chosen as one of the main topics for the Third National Congress of Ophthalmology in 1939, and a series of lectures by Miss E. E. Cass, of London, in the same year, met with fair success.

Eye surgery appears to have a special interest for the great majority of ophthalmologists in Brazil, and consequently there is a large proportion of skilled specialists who can deftly perform intracapsular extractions of cataracts and other modern surgical techniques.

Regarding eye diseases that are found in Brazil or that are common in certain sections of Brazil and in other countries of similar climatic conditions, mention should be made of Professor de Andrade's book on South American tropical ophthalmology, published in Rio in 1940. In that book a description of many ocular conditions is given, in most instances, following the author's long experience with the subject.

That edema of the lids is an early manifestation of Chagas's disease, a Brazilian trypanozomiasis, and how infection by Leishmania can cause a parenchymatous form of keratitis was mentioned in papers presented *in absentia* at the First Pan-American Congress of Ophthalmology in Cleveland, in 1940, by the same de Andrade.

In addition to the interesting data on ocular conditions in the tropical parts of Brazil that were mentioned in those papers, there are others which seem to be

less widely known, for there is hardly any mention of them in the medical literature. Fair descriptions of these eye conditions have appeared in Brazilian medical papers, but only brief résumés of them find their way into the abstract journals. Thus a few cases of obstruction of the lacrimal passages have been reported as a result of hyperostosis, a tertiary manifestation of yaws; some cases of optic neuritis in patients infected with Spirochaeta morsus muris; and a few cases of iritis due to intestinal Schistosomosis (S. mansoni), the etiology having been well established. For the treatment of this condition, as for the treatment of leishmaniasis, good results have been reported following the therapeutic use of antimonium tartrate and potassium in 2-percent solutions, 0.05 centigrams injected intravenously, daily.

A destructive ulcerous blepharitis with a predilection for the lower lid, called in the vernacular "Sapiranga" or "Gorgomi," found throughout a section of the hinterland of what is usually called the Brazilian shoulder, has been described. It generally causes ganglion reaction. De Andrade in some of his cases found that Aspergillus niger could be cultured from smears made of the secretion, and the ailment responds quite well to treatment with iodide. The study of pathologic slides failed to uncover anything typical.

The northern limits of Brazil are above the equator, and to the south it extends to a latitude corresponding to that of South Carolina. Along the coast there is abundant and regular rainfall, but in certain parts of the hinterland rain is very scant indeed. There are high plateaus with wide daily variations in the temperature. The inhabitants of the country are people whose ancestors were descendants of the Visigoths, from northern Portugal, from Germanic northern Italians or Germans proper, or from the darker Mediterranean peoples. There are those descending from the aboriginal Indians or from the imported African Negroes, and the most varied mixtures and combinations derived therefrom. The diets of these people vary from standard balanced diets through a corresponding three "M" diet to just plain Manihot utilissima flour and dried meat. Hygienic conditions range from those prevailing in a civilized city to the most primitive. All of these factors explain the varied aspects of ophthalmology that are found in Brazil, with respect to the way it is practiced, the training of ophthalmologists, the standards of research work, and the prevailing ocular diseases.

1151 Consolação

A STUDY OF THE ANISEIKONIA IN A CASE OF INCREASING UNILATERAL INDEX MYOPIA*

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Introduction

A gradual decrease in hyperopia or an increase in myopia has been observed in certain patients over 60 years of age ever since the refractive conditions of the eyes have been measured and studied.[1] It was not until the last third of the 19th century, however, that an acceptable explanation for this so-called "second sight of old people"[2] was given.

Schweigger[3] and Horner[4] expressed the opinion that because the increase in the refractive power of the eye is usually observed in cases of incipient cataract, it is due to an increase in the index of refraction of the crystalline lens, especially of the nucleus. This opinion was given an experimental basis by Zehender and Matthiesen,[5] who made actual measurements on crystalline lenses with Abbé's refractometer and found that the index of refraction of hard cataractous lenses is indeed much higher than that of normal lenses. Later research has confirmed these findings. Moreover, measurements on the crystalline lenses of humans and animals show, in general, an increase in the index of refraction with increasing age.[6] Today most authors in reporting clinical cases of increasing myopia in older people have accepted the explanation given by Schweigger and Horner.[7]

Another phenomenon of this type may be found in the so-called false lenticonus (lens with double focus) in which the increase in refraction is restricted to the embryonic nucleus. Vogt[8] states that the embryonic nucleus in such a case always shows cataractous changes, with a clear central interval in the nucleus.

These facts justify the assumption that in a patient over 60 years of age, a change in refraction accompanying nuclear sclerosis of the lens is due to an increase in the refractive power of the crystalline lens; that is, such an acquired myopia may be considered as an index myopia.

This paper reports a case of unilateral myopia of this type which was followed over a period of six years, the examinations beginning prior to the onset of the sclerosis. During this time the refraction of the right eye changed from a small hyperopia (+0.50D.) to a pronounced myopia (−3.50D.), while the refraction of the left eye remained essentially unchanged. The correction of this increasing myopia and the resulting increasing anisometropia was accompanied by a proportionate increase in aniseikonia.

The analysis of this case is important because it is such an excellent illustration of the fact that the correction of a refractive ametropia (in this case indicial) causes a change in the size of the retinal image; hence, that the correction of a refractive anisometropia is necessarily accompanied by a substantial aniseikonic error. The case is also interesting in that the recurring symptoms which accompanied the increasing anisometropia and aniseikonia were relieved by each subsequent correction of the anisometropia and aniseikonia.

* From the Dartmouth Eye Institute, Dartmouth Medical School. Read at the 350th meeting of the New England Ophthalmological Society, in Boston, on January 19, 1943.

Report of case of refractive (index) anisometropia

D. M. G., a businessman, was 60 years of age when first seen in December, 1936. There is a history of myopia in the family but to the patient's knowledge there have been no serious ocular defects. The patient was never severely ill except for a nervous breakdown at the age of 52 years, complicated by shingles. He has no diabetes. When about 10 years old the patient wore glasses for one or two years. Later he wore glasses at different times, but finding no increased comfort he usually discarded them. He has had reading glasses only since becoming presbyopic, having obtained his last correction one year prior to the examination in 1936. The patient's chief complaint was that he had always been an extremely slow reader. He also stated that frontal headaches and eye fatigue with burning and watering of the eyes occurred on use of the eyes for close work, these symptoms having become worse in the past 12 to 15 years. The patient could use his eyes comfortably for distance, although motion pictures caused some fatigue and an occasional headache.

At the time of the first examination both eyes showed no pathologic changes. The refraction was as follows: R.E. +0.50D. sph. ⩫ —0.25D. cyl. ax. 175°; L.E. +0.50D. sph. ⩫ −0.25D. cyl. ax. 80°; add for reading +2.00D. sph. The ophthalmometer measurements were: R.E. −0.62D. cyl. ax. 170°; L.E. −0.62D. cyl. ax. 20°. The muscle balance and the fusional amplitudes were within normal limits, as were the intraocular pressure and the visual fields. A measurement for aniseikonia in the horizontal and vertical meridians revealed the need for correction: ax. 90°: R.E. 0.6 percent; ax. 180°: L.E. 0.7 percent.* The combination of the refractive with the aniseikonic correction gave the patient considerable comfort in, and speeded up, his reading. Only minor changes were necessary during the years 1937 and 1938.

In December, 1939, the patient returned with a complaint of recurring discomfort at near vision and slowness in reading. A considerable increase in the refractive power of the right eye was found (−1.00D. sph. ⩫ −0.50D. cyl. ax. 170° = 20/15), while that of the left eye had remained essentially the same (+0.50D. sph. ⩫ −0.50D. cyl. ax. 95° = 20/15). Ophthalmoscopically the media of both eyes were clear; the region of both foveas showed an infiltration with fine, glittering deposits which probably were cholesterol. The slitlamp examination of the lens of the right eye revealed an increased reflex from the second layer, an increased density and a greater homogeneity in structure of the adult nucleus. The sutures could not be seen and there was a definite brownish reflex from the posterior cortex. The lens of the left eye showed similar, although considerably less marked, signs of incipient nuclear sclerosis of the crystalline lens. The aniseikonia in the horizontal and vertical meridians had changed corresponding to the change in refraction (ax. 90°: R.E. 2.0 percent; ax. 180°: R.E. 3.0 percent). The patient was given a correction for both the refractive and aniseikonic errors which proved very satisfactory.

In May, 1941, however, he returned, complaining that his reading had again slowed down. He also reported a pronounced change in his vision: With the right eye he saw better at near, with the

* The routine aniseikonic measurements referred to in this paper were made by Leo F. Madigan, Robert E. Bannon, and Wendell Triller of the Clinical Division of the Dartmouth Eye Institute.

left eye better at distance. At this time a marked increase was found in the myopia of the right eye, together with a change in the axis of the cylinder (−2.25D. sph. $\mathrel{\bigcirc\!\!\!\!=}$ −1.00D. cyl. ax. 100° = 20/20−2) while no significant change had occurred in the refraction of the left eye (+0.75D. sph. $\mathrel{\bigcirc\!\!\!\!=}$ −0.75D. cyl. ax. 90° = 20/20+2). The result of the slitlamp examination was not materially different from that in 1939; the foci in the macular regions were still present and now had the aspect of drusen of Bruch's membrane. The aniseikonia was measured while the patient was wearing the new refraction and was found to be considerably higher than before (ax. 90°: R.E. 5.0 percent; ax. 180°: R.E. 4.0 percent). The aniseikonic and the refractive errors were again corrected and the patient reported eye comfort until the spring of 1942.

In July, 1942, the patient once more reported pronounced discomfort at near vision and slowing down of reading. He had noticed further significant changes in his visual acuity; the difference between the two eyes was now even more marked. Occasionally flashes had appeared before his eyes, lasting only a short while, but he had otherwise enjoyed good health and had had no headaches or other symptoms of ocular discomfort. A further increase in the refraction of the right eye only was found (R.E. −3.50D. sph. $\mathrel{\bigcirc\!\!\!\!=}$ −1.25D. cyl. ax. 95° = 20/25−3; L.E. +0.75D. sph. $\mathrel{\bigcirc\!\!\!\!=}$ −0.50D. cyl. ax. = 20/15−2), together with a corresponding increase in the aniseikonia (ax. 90°: R.E. 9.0 percent; ax. 180°: R.E.

TABLE 1

TABLE SHOWING OCULAR REFRACTION AND THE ACCOMPANYING ANISEIKONIA AS MEASURED AND COMPUTED FOR A PATIENT WITH INCREASING UNILATERAL INDEX MYOPIA STUDIED OVER A PERIOD OF SIX YEARS

(1)	(2)	(3)	(4)	(5)		(6)	(7)	(8)	(9)	(10)
					Measured Aniseikonia			Computed Aniseikonia—%		
Examination Date	Refraction	Indicial Myopia	Lens	Instrument	Meridian	Percent	Total	Due to Indicial Myopia	Due to Corneal Astigmat.	Due to the Lenses
12-22-36	R +0.50 −0.25 ×175 L +0.50 −0.25 × 80	None	T.C.	O.E.	H V	L 0.6±0.1 R 0.7±0.5	L 0.5 R 0.5	None	L 0.4 R 0.4	L 0.1 R 0.1
2- 2-37	R +0.50 −0.25 ×175 L +0.50 −0.25 × 80	None	T.C.	O.E.	H V	0.0±0.7 R 1.2±0.5	L 0.5 R 0.5	None	L 0.4 R 0.4	L 0.1 R 0.1
8-22-38	R +0.25 −0.50 ×180 L +0.75 −0.25 × 90	−0.25D.	T.C.	O.E.	H V	R 0.4±1.0 R 1.2±1.5	R 0.1 R 1.8	R 0.7	L 0.5 R 0.9	L 0.1 R 0.2
12-27-39	R −1.00 −0.50 ×170 L +0.50 −0.50 × 95	−1.50D.	T.C.	O.E.	H V	R 2.0±2.0 R 3.0±1.0	R 1.6 R 3.9	R 2.7	L 0.9 R 0.9	L 0.2 R 0.2
5-28-41	R −2.25 −1.00 ×100 L +0.75 −0.75 × 90	−2.75D.	T.C.	O.E.	H V	R 5.0±1.0 R 4.0±1.0	R 4.7 R 4.2	R 4.2	R 0.4 0.0	R 0.1 0.0
	Same		Spec.	S.E.	H V	R 4.4±0.5 R 5.2±0.5	R 5.1 R 4.8	R 4.1	R 0.4 R 0.1	R 0.6 R 0.6
7 3-42	R −3.50 −1.25 × 95 L +0.75 −0.50 × 95	−4.00D.	T.C.	O.E.	H V	R 9.0±1.0 R 8.0±1.0	R 9.0 R 7.3	R 7.3	R 1.4 0.0	R 0.3 0.0
	Same		Spec.	O.E.	H V	R 9.0±1.0 R 8.0±1.0	R 9.4 R 8.0	R 7.0	R 1.5 R 0.1	R 0.9 R 0.9
	Same		Spec.	S.E.	H V	R 8.2±0.3 R 7.5±0.5	R 9.4 R 8.0	R 7.0	R 1.5 R 0.1	R 0.9 R 0.9

T.C. Trial case test lenses.
Spec. Spectacles.
O.E. Standard ophthalmo-eikonometer.
S.E. Space eikonometer.
H Horizontal meridian, axis 90°.
V Vertical meridian, axis 180°.

8.0 percent). The condition of the fundi was the same as on previous visits; the nucleus of the lens of the right eye presented an even more marked sclerosis than before, but the cortex was still clear.

DISCUSSION OF THE CASE

The various measurements of refraction and aniseikonia as well as the results of a calculation to be described are summarized in table 1.

The refractive measurements are tabu-

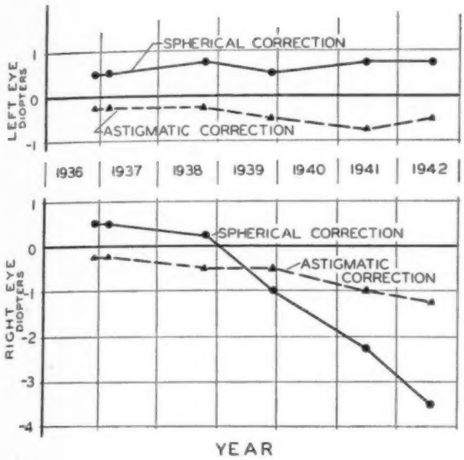

Fig. 1 (Burian and Ogle). Graph of the spherical and astigmatic refractive errors of patient during a period of six years. The increase in the myopia of the right eye is strikingly illustrated. This myopia is due to the increase in index of refraction of the crystalline lens associated with a nuclear sclerosis.

lated in column 2 of table 1. They are also represented graphically in figure 1, which illustrates strikingly the increased myopia in the right eye. It would appear from this figure that the sclerosis of the nucleus of the crystalline lens began rather abruptly in 1938, and that it has increased since then at a nearly constant rate.

In column 6 of table 1 are tabulated measurements of the aniseikonia in the horizontal (ax. 90°) and the vertical (ax. 180°) meridians. The entries are the percent magnifications needed before the eye indicated to equalize the sizes of the ocular images in the meridian specified.

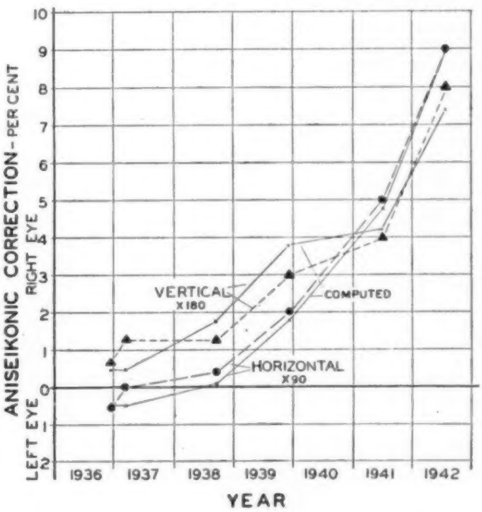

Fig. 2 (Burian and Ogle). Graph of the aniseikonia of a patient with increasing unilateral myopia over a period of six years, showing the marked increase that accompanies the progress of the myopia. The solid lines are the calculated values.

It should be noted that in 1941 and 1942 the aniseikonic measurements were made on the new space-eikonometer as well as on the standard ophthalmo-eikonometer.* Column 4 indicates whether trial-case test lenses or spectacle lenses were used during the aniseikonic examination, and column 5 the particular instrument used in the examination. These aniseikonic measurements are graphically shown in figure 2. It should be pointed out here that the present study was not concerned with the problem of the aniseikonia resulting from the uncorrected unilateral myopia itself but only with the anisekonia result-

* A definite correlation between the measurements on these two instruments has been established.[8] For a more complete description of the relationship between binocular spatial localization and anomalous ocular image differences the reader is referred to a paper to be published by Prof. A. Ames, Jr., entitled "Binocular spatial localization and anomalous ocular image differences."

ing after the myopia had been corrected.

Inspection of the table and of figures 1 and 2 relating to the refractive and aniseikonic measurements, shows that a rapid increase in the relative difference in the size of the ocular images of the two eyes occurs with the increase in the myopia of the right eye. This increase in aniseikonia is in the expected direction: The size of the image of the right eye, when the myopia is corrected, decreases relative to the size of the image of the left eye, and therefore an increased magnification of the image of the right eye is necessary to equalize the size of the images of the two eyes.

This case is especially suitable for the quantitative demonstration of the fact that the correction of a refractive ametropia results in a change in the size of the retinal image, because the increasing myopia occurs only in one eye, the refractive error of the other remaining constant, and because the origin of the myopia is known to be due to an effective increase in refractive index of the crystalline lens. These two facts make it possible to calculate the expected increase in aniseikonia which can then be compared with the measured increase as the myopia increases.

If the ametropia in the right eye were not entirely refractive (indicial), but axial in nature, this computation could not be made, since Knapp's law would apply. This law states that if the second principal plane of a correcting ophthalmic lens coincides with the anterior focal point of the ametropic eye, the size of the retinal image is the same as though the eye were emmetropic.*

The calculation of the aniseikonia

The calculation of the aniseikonia for each examination involves three parts:† The determination of (A) that part of the image size difference due to the correction of the index myopia; (B) that part due to the correction of the corneal astigmatism; and (C) the modifications in the differences due to the optical properties of the particular spectacles or trial-case test lenses used in the aniseikonic examinations.

The results of these calculations are shown in columns 7-10 of table 1. The measured aniseikonia and that computed are given in columns 6 and 7, which are placed side by side to facilitate comparison.

(A) Index myopia. The calculations of the change in the size of the retinal image were simple computations of paraxial rays through a schematic eye and a correcting lens. Since only the change in the size of the retinal image corresponding to an increase in the index of refraction of the crystalline lens is to be computed, the particular schematic eye selected is not important; the differences in the calculations that would occur with the use of a different eye would be of a second order. The scheme of calculation and the constants of the schematic eye used (which is a modification of the Tscherning eye[11] in which the substance of the crystalline lens is considered homogeneous‡) are shown in figure 3. The con-

* The Knapp (and Giraud-Teulon) law.[10] One of us (K. N. O.) will consider this problem more generally in a forthcoming publication.

† Strictly speaking only the aniseikonia due to the myopic condition can be calculated since the nature of the previous refractive errors is not known. However, this fact proved to have little effect upon the problem since the aniseikonia measured prior to the onset of the myopia is small and is accounted for by the astigmatic corrections.

‡ The stratified nature of the lens and the differences in the index of refraction of its various layers can be disregarded; for these calculations the index considered is the effective index of refraction of the crystalline lens as a whole.

stants are so selected that with a normal refractive index of the crystalline lens (no correcting lens is necessary) the image of an object at a distance of 6 meters from the eye will be focused on the retina. The basic magnification, M_o, of the retinal image for the normal eye as compared to the size of an object at 6 meters is first determined from $M_o = \frac{i_o}{O} = \frac{\pi U}{\pi V}$, where i_o is the size of the retinal image in the normal schematic eye and O is the size of the object and where πU and πV are the products of the tracing a paraxial ray from the retina toward the object, the power (diopters) of this correcting lens can be determined. The magnification of the retinal image relative to the object is then computed from M = i/0, where i is the size of the image of the corrected ametropic eye and 0 is again the size of the object. The ratio, R, of this latter magnification, M, to the normal basic magnification, M_o, existing before the change in index, is then determined to give the magnification (in this case a reduction) of the size of the image, caused by the increase in the index

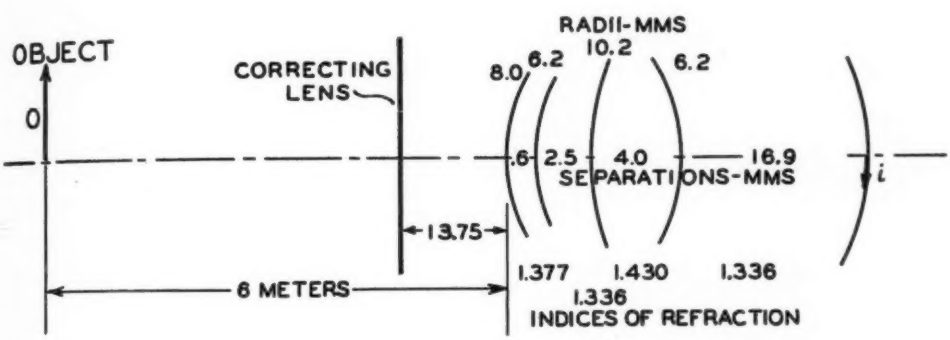

Fig. 3 (Burian and Ogle). The constants of the (modified Tscherning) schematic eye used in the calculation of the expected aniseikonia that accompanied the increased index myopia.

successive object and image vergences at the successive refractive surfaces of the eye.

The index of refraction of the crystalline lens is then given a slightly higher value, which results in a myopia that is to be corrected by an ophthalmic lens before the eye. For this calculation the correcting lens is so located that its second principal plane will be at a distance of 13.75 mm. from the cornea of the schematic eye. This distance corresponds to the distance from the eye of the trial-case test lenses in the eikonometer. By of refraction and by the lens needed to correct the resultant myopia. That is, $R = M/M_o = i/i_o$. This computation is repeated for further increases in the effective index of refraction of the crystalline lens.

Figure 4 illustrates the results of these computations showing (1) the dioptric power of the correcting lens and (2) the corresponding change in magnification in percent for an increase in the effective index of refraction of the crystalline lens of from 0.01 to 0.04.* Using

* Hess (see ref. 1) states that the decrease of the effective index of refraction of the crystalline lens by 0.01 (for example, from 1.43 to 1.42) reduces the refractive power of the eye by approximately 2D.

this graph, the diminution of the retinal image that might be expected theoretically in the right eye in the case under discussion can be interpolated.* The theoretical magnification needed to equalize the images of the two eyes, corresponding to the spherical myopia of the right eye, can then be read from the same graph.

The results of this part of the calculation are given in column 8 of table 1, and are indicated in figure 4 by the vertical lines corresponding to the dates of the examination. These values could be used alone to show the relation between the change in aniseikonia that accompanies the progressive index myopia, for they account for the greater part of that change.

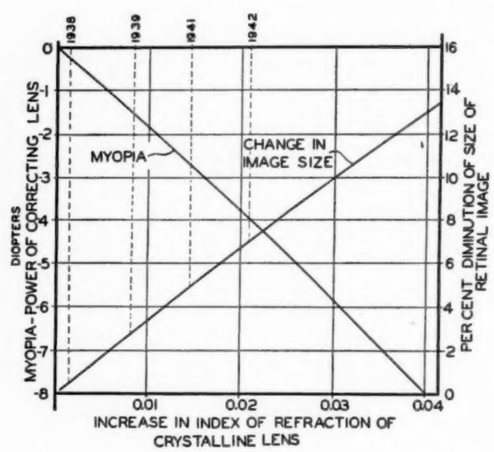

Fig. 4 (Burian and Ogle). Graph showing the theoretical myopia that accompanies an increase in the index of refraction of the crystalline lens, and the theoretical change in the size of the retinal image that would be expected upon the correction of the myopia.

(B) Astigmatic correction. The calculated values will be more accurate if the part of the aniseikonia due to the astigmatic correction is determined, since astigmatic ametropia is always a refractive ametropia. This part of the total aniseikonia proves to be small compared with that due to the index myopia. Again placing the correcting lens at 13.75 mm. from the cornea, and taking into account the keratometric findings, one can calculate the change in the retinal-image size quite accurately for small astigmatic errors from the relation $M = Dh/10$, where M is the percent change in the retinal-image size, D the power in diopters of the astigmatic correction, and h the distance in millimeters of the correcting lens from the site of the astigmatic error (13.75 if entirely corneal; approximately 19.0 if entirely lenticular).

The results of these calculations for the vertical and horizontal meridians for the two eyes are given in column 9 of table 1.

(C) Corrective lenses. Both the afore-described calculations were made for infinitely thin correcting lenses at a specified distance from the eyes. The actual lenses worn during the aniseikonic examination, due to their flexure, thickness, and distances from the eyes, would modify these calculations. Knowing the physical dimensions of the lenses used in each measurement, the appropriate corrective modifications can readily be made.[12] These modifications proved to be relatively small when trial-case test lenses were used, and only slightly larger when spectacles were worn by the patient.

The magnitude of these modifications in terms of the aniseikonic correction is given in column 10 of table 1.

The sum of these three calculated

* It is possible, though more difficult, to solve the paraxial ray equations directly for the effective index of refraction of the crystalline lens corresponding to a given myopic correction. The method given above, however, seemed to illustrate more clearly the principle involved.

values is the theoretical aniseikonia to be expected on the basis of the known refractive ametropia and the optical properties of the correcting lenses. These are given in table 1, column 7, which is placed beside the column giving actual aniseikonic measurements. The results of the calculations are also shown graphically in figure 2 by the solid lines. The close agreement between the measured and the computed amount of aniseikonia is evident from the table and the graph. Attention is also called to the fact that the agreement is true for measurements obtained both upon the new space-eikonometer as well as the standard eikonometer.

This agreement substantiates the original hypothesis, made on the basis of the clinical evidence, that the myopia in this case was due to an increase in the index of refraction of the crystalline lens. A particular case of nuclear sclerosis of an eye studied by Heine[13] is of interest in this connection for he was able to measure the index of refraction of the crystalline lens upon its subsequent removal. The eye was 4 to 5 diopters myopic and Heine measured the effective index as 1.452. This figure is identical with that computed for the case under discussion if it is assumed that the effective index of refraction of the crystalline lens of the right eye was normal—namely, 1.43—prior to the onset of the nuclear sclerosis. A myopia of 4 diopters would correspond to an index of 1.453 (see fig. 4).

The analysis of the case reported in this paper demonstrates the fact that a refractive anisometropia, when corrected, is accompanied by a refractive aniseikonia, but it must again be emphasized that this does not mean that a refractive aniseikonia will be present in every case of corrected anisometropia.[14] It occurs only when the ametropia is, at least in part, due to refractive causes; that is, the ametropia is not axial in origin. This statement may be illustrated by the report of a second patient.

Report of a case of axial anisometropia

Mr. M. G., a student, aged 22 years, without significant ocular family history and in good general health, complained of eyestrain in doing close work. Examination revealed that both eyes were externally normal and the media clear. The fundus of the right eye showed no pathologic changes. In the fundus of the left eye there were signs of myopic stretching; such as, a temporal crescent and straightened blood vessels at the posterior pole. The refraction and visual acuity were as follows: R.E. +0.50D. sph. ⊂⊃ −0.50D. cyl. ax. 135° = 20/15; L.E. −2.75D. sph. ⊂⊃ −0.50D. cyl. ax. 105° = 20/15. There was no significant muscle imbalance, and the patient had normal stereopsis with the refractive correction. The examination for aniseikonia gave these results: On the standard eikonometer: (distance vision) ax. 90°: L.E. 0.2% ± 0.7%; ax. 180°: 0% ± 0.5%. On the space-eikonometer: ax. 90°: R.E. 0.4% ± 0.5%; ax. 180°: L.E. 0.4% ± 0.5%.

Because of the absence of a significant amount of aniseikonia we were forced to conclude that in this case the refractive error of the left eye was axial in nature, a conclusion which was corroborated by the clinical appearance of its fundus. Thus, in general, *a priori* conclusions as to the presence of aniseikonia in cases of anisometropia cannot be made.

Differential eye movements

In view of a suggestion[15] that the measurements obtained with the standard eikonometer, particularly in cases of anisometropia, may in fact be due to differential eye movements, caused by the corrective

lenses, it appeared to be of interest to determine the magnitude of the differential prismatic effect in these cases.

The angle through which the eye has to turn from its primary position to look at an object above, below, to the right, or left of the fixation point is larger when the object is seen through a positive lens and smaller when it is seen through a negative lens than it is when seen without a lens. These differences are due to a prismatic effect of the lenses for objects

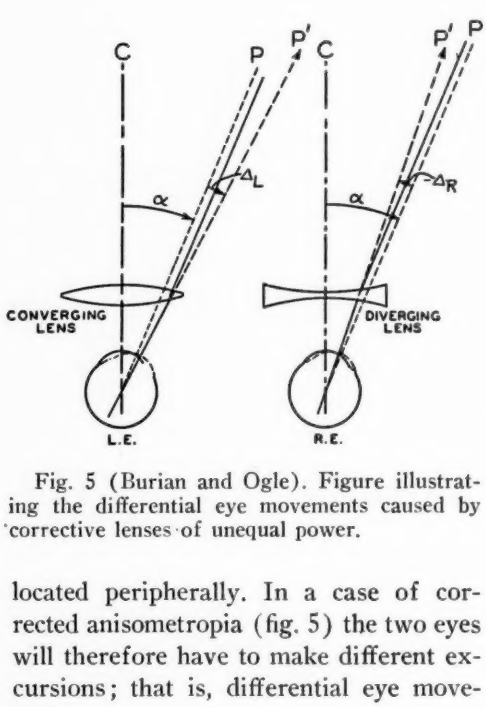

Fig. 5 (Burian and Ogle). Figure illustrating the differential eye movements caused by corrective lenses of unequal power.

located peripherally. In a case of corrected anisometropia (fig. 5) the two eyes will therefore have to make different excursions; that is, differential eye movements.

The magnitude of the prismatic effect depends upon the angular distance of the peripheral object from the fixation point, the power and physical dimensions of the correcting lens, and the distance of this lens from the center of rotation of the eye. For a thin lens this prismatic effect is given with sufficient accuracy by $\Delta = Dd\alpha$, where Δ is the angular deviation, D is the power of the lens in diopters, d is the distance of the lens from the center of rotation of the eye in meters, and α the angular distance of the point peripherally located. The percent increase or decrease of the angle through which the eye must turn (which is equivalent to the percent angular magnification of the lens, referred to the center of rotation of the eye) would be $\frac{\Delta}{\alpha}\% = 100$ Dd.* This relation has the advantage of expressing the prismatic effect as a single quantity for all angles.

Using this formula, and allowing for the flexures and thicknesses of correcting lenses, the magnitudes of the differential eye movements in percentage were computed for each of the measured refractive corrections of the first patient (D. M. G.) They are given in table 2 and shown graphically in figure 6. For comparison, the measured amounts of aniseikonia are included in the table and

TABLE 2

THE COMPUTED PERCENT DIFFERENTIAL EYE MOVEMENTS FOR THE CORRECTED ANISOMETROPIA OF D. M. G. AND THE MEASUREMENTS FROM THE STANDARD EIKONOMETER, FOR THE HORIZONTAL AND VERTICAL MERIDIANS.

Date of Examination	Meridian	Measured Aniseikonia	Differential Eye Movements
1936	H	0.0–L 0.6%	L 0.8%
	V	R 0.7–R 1.2%	R 0.8%
1938	H	R 0.4%	R 0.6%
	V	R 1.2%	R 2.9%
1939	H	R 2.0%	R 2.5%
	V	R 3.0%	R 5.5%
1941	H	R 5.0%	R 8.3%
	V	R 4.0%	R 7.9%
1942	H	R 9.0%	R 13.2%
	V	R 8.0%	R 11.7%

* The average distance of center of rotation behind the cornea of the eye is taken to be 13.6 mm. (cf. Duke-Elder, W. S.: Textbook of ophthalmology. London, Kimpton, 1932, v. 1, pp. 582-583).

the graph. From a study of these results, it is evident that the values of the aniseikonic measurements, with which the computed aniseikonia agreed closely, are generally smaller than the theoretical values for the differential ocular movements. A similarity in the trend of the differential ocular movements with the aniseikonia in this case is to be ex-

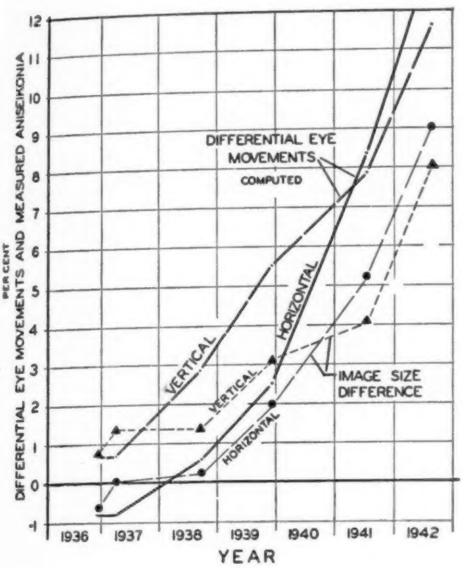

Fig. 6 (Burian and Ogle). Graph showing the theoretical magnitude of the differential ocular movements that would occur in the patient with increasing unilateral lenticular index myopia, and the aniseikonia measured on the standard eikonometer.

pected, for the refractive error in one eye is small, whereas that of the other increases and has its origin at a place in the eye—namely, the center of the crystalline lens—which is not far distant from the center of rotation of the eye itself. The difference between the magnitude of the differential ocular movements and the aniseikonia would be larger if the origin of the ametropia in the right eye were entirely corneal. The differences, however, between the measurements and the calculated differential ocular movements are so significant, that one may safely conclude that in this case the measurements obtained on the standard eikonometer were measurements of the difference in the size of the ocular images and not of the differential eye movements caused by the anisometropic corrections.

The evidence for this conclusion is given additional weight by the fact that the measurements on the second patient (M. G.) showed no significant image-size difference either with the standard eikonometer or with the space-eikonometer. If the differential eye movements that would be caused by the corrective lenses for M. G.'s anisometropia would have influenced the measurements, an angular percent difference of nearly 2.7 percent would have manifested itself.

It must again be emphasized that the measurements obtained on the standard eikonometer are in agreement with those obtained on the space-eikonometer where eye movements play no part in the binocular spatial localization on which that test* is based.

Conclusions

1. The clinical and analytic study of a case of increasing unilateral myopia due to nuclear sclerosis of the crystalline lens demonstrates the fact that refractive anisometropia is accompanied by aniseikonia.

2. This case is contrasted with one having a similar degree of anisometropia not due to a unilateral nuclear sclerosis of the crystalline lens. No significant image-size difference was measured in the second case and the clinical evidence tended to show the myopia to be axial in nature. Thus it was pointed out that no *a priori* conclusions can be made as to the aniseikonia present in any given case of anisometropia.

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* See footnote, page 483.

3. Finally, it is shown that the measurements obtained with the standard eikonometer (and space-eikonometer) are not measurements of the differential ocular movements caused by the prismatic effect of the lenses correcting the anisometropia.

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SEVERE KERATO-IRITIS DUE TO BRUCELLOSIS: SUCCESSFUL TREATMENT WITH BRUCELLA ABORTUS VACCINE

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Saint Louis

When confronted with an inflammatory affection of the cornea, iris, ciliary body, choroid, retina, and optic nerve, the alert ophthalmologist will at once begin searching for a local or general pathologic condition which might conceivably be the true cause of the ocular malady.

Guided by the ophthalmologist, the diagnostician will usually be asked to examine the patient with a view to the following possible diagnoses: Syphilis, tuberculosis, focal infections (sinusitis, tonsillar and dental infection, cholecystitis, chronic appendicitis, chronic prostatitis), and (in the female) pelvic inflammation and infection. Evidences of chronic nephritis and mild or severe diabetes may be uncovered. If a critical diagnostic survey reveals evidence of general disorders, or foci of infection, the therapeutic attack is at once directed toward alleviation or, if possible, elimination of the discovered abnormalities. The ophthalmologist may consider himself fortunate if the investigation has brought to light some well-defined general or local pathologic condition which, when ameliorated or eliminated, is followed by such prompt improvement in the ocular condition as to justify the assumption of a causal relationship.

There is, however, another side to the picture. The diagnostician may have found your patient generally sound, free from allergies or evidence of vitamin deficiencies. In his endeavor to cover the entire field, he has probably called to his aid the otolaryngologist, the urologist, the gynecologist, the dentist, and the laboratory man. The otolaryngologist may state that the tonsils have been cleanly removed and that there is no X-ray nor clinical evidence of sinus infection. He may also vouchsafe the information that there is no evidence of chronic middle-ear trouble or infection lurking in the mastoid.

The urologist and gynecologist may deny evidence of pathologic changes in their respective fields. The dentist, backed up by radiographs of the teeth, may report, that, save for a few small fillings in vital teeth and a tendency to recession of the gums, there is nothing abnormal. He will be especially positive in his opinion that there is no clinical nor radiographic evidence of resorption of the roots or apical abscesses. Finally, the laboratory man reports a normal blood picture, negative blood cultures, and a negative blood Wassermann test.

When all the reports are in, the ophthalmologist finds that nothing has emerged that would warrant the institution of any particular therapy. Many colleagues will agree that such a baffling situation is by no means rare. There is no clear indication as to any particular therapy, yet the urge to do something is strong. This leads to therapeutic experimentation, in the hope (often vain) that, somehow, something will be hit upon that will benefit the ocular condition.

I assume that the foregoing description comprises what ophthalmologists generally would accept as a fairly complete survey. It is my considered opinion that one important disease has been overlooked; namely, undulant fever or brucellosis. Since the publication of my paper,[1] I have had no occasion to alter my conviction that brucellosis is the true etiologic factor

in many inflammatory ocular diseases.

This disease may be ushered in by an acute episode—high fever, headache, muscular aching, and prostration. A correct diagnosis is infrequent. Perhaps typhoid fever and malaria are considered, or even acute miliary tuberculosis; these have to be ruled out because of the lack of confirmatory findings. With or without treatment, the fever abates. The disease, however, has not run its course; it has only changed into the chronic form. The patient never feels entirely well but is able to carry on his usual occupation. Then may come days wherein he suffers from ill-defined muscular pains and stiffness in the joints accompanied by mental depression and inability to perform consecutive mental tasks. During these periods the patient feels toxic or, as one of mine expressed it: "I feel just as though I had a hangover." This period may be afebrile or it may be accompanied by a low afternoon fever.

There is good reason to believe that with some patients the initial acute symptoms may be lacking and that from the beginning the symptomatology is that of the chronic form.

If the clinician, his suspicions aroused, seeks confirmatory evidence of the existence of brucellosis from his hospital laboratory, it is highly probable that the laboratory will carry out the one test that has the least value in uncovering chronic brucellosis; namely, the agglutination test. It is well known that this test will be negative in 90 percent or more of proved cases of brucellosis. It is astonishing that, realizing the difficulties and uncertainties of the clinical diagnosis, the laboratories should not make use of the skin test and the valuable and confirmatory opsonocytophagic test. If the agglutination test alone has been made, a negative report is meaningless and misleading.

Case report*

Miss P. N., aged 24 years, appeared at the Eye Clinic of the Jewish Hospital on April 13, 1938. Vision, R.E., was 20/20 —3; L.E., 20/40+2. The patient stated that her left eye had been inflamed for a week. The right eye showed no abnormality. The left eye showed circumcorneal injection; the tension was normal to full; the anterior chamber was normal. There were increased aqueous flare, 10 cells per high-power field, and many dense, broad posterior synechiae. The lens was clear, the fundus normal, through a small pupil.

Under the influence of atropine 1 percent and suprarenin ointment 1 percent the pupil dilated irregularly. One-percent atropine was prescribed. On April 18th, there remaining a number of firm synechiae, a subconjunctival injection of atropine, cocaine and adrenalin was made. In spite of these very active measures to promote mydriasis, numerous synechiae remained. On May 2, 1938, it was noted for the first time that the upper nasal quadrant of the cornea showed some deep vascularization.

The patient was referred to the medical clinic, where it was noted that her mental state was deficient. Acne was observed on the face and back. Urinalysis was negative. The red blood-cell count was 4,390,000; hemoglobin, 92 percent. The white blood-cells count was 11,300:

	Percent
Eosinophiles	2
Stabs	3
Segmenteds	67
Lymphocytes	27
Monocytes	1

The basal metabolic rate was —9. Tu-

* For the clinical notes covering the observation and treatment of Miss P. N. at the Jewish Hospital Eye Clinic, I am indebted to Dr. Daniel Bisno, chief of the Eye Clinic in this Hospital.

berculin tests were negative, as was the Wassermann (blood). Both tympanic membranes were scarred and retracted. Hearing was normal. The tonsils and adenoids had been removed. Dental radiographs showed evidence of peridental infection in the molar area of the right second bicuspid. There was no evidence of canal fillings or apical abscesses.

Gynecologic examination revealed chronic endocervicitis of undetermined origin. The report from the chest clinic revealed no abnormal lung findings other than calcified hilar glands.*

SUGAR DETERMINATION

	Fasting	Hour 1st	2d	3d	4th
Blood	89	143	100	79	98
Urine	Neg	Neg	Neg	Neg	Neg

Diastase, 83; cholesterol, 238.

During one period of hospitalization, a lumbar puncture was made. The spinal fluid was not under pressure and was reported to be normal.

Two months after the first observation the right eye for the first time showed circumcorneal congestion. By biomicroscopy, cells were found in the anterior chamber and there were fine dustlike deposits on Descemet's membrane. The pupil of the right eye was irregular. The left eye now showed definite infiltration in the deeper layers of the cornea, with increased vascularity. It was noted that the pupil was beginning to fill with exudate.

The patient was hospitalized from June 24 to July 17, 1938. During this period typhoid-paratyphoid vaccine was administered intravenously: the first dose, 25,000,000; second dose, 50,000,000 third dose, 100,000,000. Following the first and second injections there were good febrile reactions.

On July 20th, vision R.E. was 20/20—1; L.E., 20/100.

With the biomicroscope there were found 10 cells to the high-power field in each eye. It was noted that there was beginning deep vascularization of the cornea of the right eye. The eyeground was, however, still visible and appeared to be normal. The fundus of the left eye was obscured by corneal opacities, posterior synechiae, and fibrinous deposits on the anterior lens capsule. Local treatment consisted of atropine in both eyes three times a day. Internally, the patient was given sodium salicylate, gr. 10, three times a day.

On September 12, 1938, an intradermal tuberculin test, first with 0.1 mg. and later with 1 mg. O. T., gave negative results locally, focally, and constitutionally.

On October 21, 1938, vision in the right eye remained 20/20 in spite of the continuance of the iritis. Vision in the left eye had diminished to light perception. On account of atropine irritation, hyoscine was substituted for atropine.

On November 4, 1938, vision R.E., was 20/50 plus. The decrease in vision of the right eye was ascribed to the increased infiltration and vascularity of the cornea.

On December 15, 1938, the patient had a transfusion of 450 c.c. of citrated blood. Two weeks later it was noted that the eyes were slightly better; the improvement being ascribed to the transfusion. This led to the administration of a second transfusion on January 22, 1939.

On February 22, 1939, the sedimentation rate was 27; PSP, first hour, 45; second hour, 15.

On February 24, 1939, agglutination tests for Brucella abortus and Brucella melitensis were made. This resulted in a negative reaction for Brucella abortus in

* Hilar, peribronchial thickening and fibrosis have been noted frequently in chronic brucellosis. These X-ray findings should not, therefore, be regarded as indubitable evidence of tuberculosis when tuberculin tests are negative.[2]

all dilutions from 1:40 to 1:320. For Brucella melitensis there was a positive reaction for the 1:40 dilution; negative for all the others. The skin test and opsonocytophagic tests were not made.

On March 2, 1939, sulphanilamide, gr. 10, four times a day, was prescribed.

The patient was again hospitalized from March 25 to April 14, 1939, and during this period received six treatments in the Kettering hypertherm. The exposures lasted four hours and the rectal temperatures reached 105 to 106°F.

On April 26, 1939, vision R.E., was 20/20—4; L.E., the ability to perceive hand motions only.

From May 4 to June 2, 1939, gold therapy by intragluteal injection was instituted as follows: myochrisine (sol. gold sodium thiomalate) 0.025 gm. A few days later this was repeated in the same dose. The three following doses were, respectively, 0.050 gm., 0.075 gm., and 0.01 gm. No favorable effect was noted following this therapy.

On June 9, 1939, neoarsphenamin, 0.15 gm., was administered intravenously.

On June 16, 1939, the notes stated: "Cornea seems cloudier; there is an increase in the aqueous flare and there are also more vessels penetrating the cornea."

On June 22, 1939, the patient was seen in consultation by several of the oculists of the Washington University Eye Clinic. The consensus of opinion was that the trouble was some form of tuberculous infection or sensitivity. The prognosis was considered serious and treatment along general lines—fresh air, complete rest, adequate diet—was advised.

From July 10 to August 31, 1939, new acneiform lesions appeared on the face and arms.

On September 29, 1939, the patient was given Armour's thyroid, gr. 1, three times a day.

Between October 1, 1939, and January 17, 1940, she received 10 treatments of short-wave diathermy to each eye. These treatments were of 20 minutes' duration. Toward the end of this period she received three radium treatments (beta radiation).

On January 17, 1940, vision of the right eye had been reduced to 4/100. In the meantime the corneal infiltrates were projecting in from all parts of the cornea. On this date it was noted that the iris of the left eye was bombé. The tension was: right, 11 mm. Hg (Schiötz); left 47 mm. Local treatment consisted of scopolamine 0.25 percent to the right eye; eserine, 0.5 percent to the left eye—three times a day.

On March 13, 1940, vision, R.E., sufficed only to count fingers at 18 inches; L.E., light perception.

The patient was hospitalized from March 20 to March 31, 1940, during which time she received four intravenous typhoid-paratyphoid injections: respectively, 50,000,000, 100,000,000, 125,000,000, and 200,000,000.

On April 12 and 14, 1940, she received general bodily treatment with ultraviolet. In the meantime the synechiae in the right eye failed to break and did not yield to neosynephrin, a 10-percent emulsion.

The patient was again hospitalized from April 24, to May 11, 1940. Another course of typhoid-paratyphoid therapy was carried out. This was followed up by one session in the Kettering hypertherm.

On May 23, 1940, vision, R.E., was ability to detect hand motion at one foot.

In June, July, and early August, the patient received riboflavin, at first 1 mg. daily, then increased to 10 mg. daily. No improvement was noted in the eyes.

From August 11 to September 7, 1940, the patient received a short course of O. T. tuberculin, dosage beginning at 1/1,000 mg. The tuberculin was continued from September 14, 1940, to Febru-

ary 14, 1941, the final dosage reaching 1/10 mg.

On February 20, 1941, the patient was transferred to the National Jewish Hospital in Denver, Colorado, where she remained until September of 1941. Through the courtesy of Dr. Albert Guggenheim, acting Medical Director of the National Jewish Hospital, I received the following "Summary on Discharge" (slightly abridged).

P. N., a white female, was admitted to the National Jewish Hospital on 2-28-41 at the age of 26. . . . Family history was not contributory. Personal history essentially normal.

Past medical history: Measles, chickenpox in childhood.

Present illness: Apparently fluid (?) developed in left eye in 1938. Eye was treated first by local applications. . . . Was then referred to Jewish Hospital in St. Louis where she was thoroughly treated. Diagnosis at that time was kerato-iritis, probably tuberculous. . . .

Physical examination on admission was essentially negative, except for eye findings. The following was noted by eye consultant. "Right eye: Ciliary injection. In the center of the cornea are numerous deep and superficial scars following keratitis. In the upper part of the scar are fresh infiltrations. Deep and superficial blood vessels cross the limbus and reach to the middle of the cornea. No precipitates. The aqueous is clear, the iris atrophic, and shows no signs of present inflammation. Numerous old posterior synechiae so that the pupil does not dilate with atropine. Lens apparently clear. Fundus not visible on account of corneal scars and the narrow pupil. Tension normal. Vision: R., counts fingers at 1½ m. L., light perception. Old scars in the cornea, anterior chamber very shallow, iris atrophic, pupil very narrow closed by synechiae. Lens slightly opaque. Fundus not visible. Tension now normal; was markedly increased. . . ."

Tuberculin test was positive with .1 mg. tuberculin. Chest X ray revealed bilateral calcification in hili—otherwise negative. Subsequent X rays showed no essential changes. . . . Sedimentation rate 20 mm. per hour. Urine negative. Patient had no sputum and eight gastric lavages were negative for tubercle bacilli. One guinea pig inoculated with lavage, done at admission, was negative. No positive reported. One culture of gastric lavage reported negative.

Course in Hospital: As recommended by eye consultant, tuberculin therapy in gradually increasing doses beginning with 0.1 c.c. of 100,000,000 was instituted and continued two times a week by intradermal injections. Dose shortly before discharge was 5 c.c. of 1:100,000. No local or systemic reaction. Three mg. riboflavin were given daily and 2 drops scopolamine ¼% at bedtime in right eye. Temperature and pulse remained normal throughout.

Patient was seen frequently by an ophthalmologist who reported no sustained substantial improvement. At her own request patient was discharged on August 8, 1941. . . .

Final diagnosis: Bilateral keratitis—tuberculous. Left iritis with iris bombé.

On October 5, 1941, the patient returned to the Jewish Hospital. At this time vision R.E. enabled her to see hand motion at two feet; L.E., light perception.

In November, 1941, the patient spent a few days in the Hospital for two sessions of the Kettering hypertherm.

This patient came under my observation on November 26, 1941.

History. Patient had a normal birth, and was nursed by the mother. When the child was three weeks old, the family moved to the country and lived there seven months; shortly after returning to Saint Louis the child was weaned. Milk was then obtained from a neighbor who owned a cow. There was no attempt at pasteurization or sterilization, and the raw milk was fed to the child. This routine continued for eight or nine years.

Examination. The patient was a small, decidedly plump individual. It was quite evident that she was subnormal mentally. There were numerous acne pustules on the face and neck. The hair was thick and coarse; and the hair line extended low on the forehead. The patient was uncomplaining and placid. Throughout the entire course of my observation and treatment she has remained happy and contented; in fact, has tended to be over optimistic. This state of mind was definitely surprising in view of the fact that she had undergone prolonged local and general treatment, with an uninterrupted tendency toward deterioration of vision.

Ocular examination and treatment. Both eyes showed marked ciliary congestion and epiphora. Vision R.E., enabled her to see fingers at one foot. The tension was 19 mm. Hg. (new Schiötz). The cornea of the right eye showed deep vascularization, the vessels coming in mainly from the nasal side and below. There was a central opacity. The clearest portion of the cornea was above. The iris was fairly well seen, contained numerous synechiae; the anterior chamber was of normal depth. The ophthalmoscope yielded a dull red reflex, no details.

Vision L.E., was light perception at 3 feet; faulty projection. The tension was 38 mm. Hg. There was a deep central corneal infiltrate extending nasally. The infiltrate was deeply vascularized. The pupil was small, vertically oval, the iris margin adherent to a pupillary membrane. The anterior chamber was all but obliterated, the iris tissue being ballooned—iris bombé.

The patient was sent to the laboratory for a serologic investigation for brucellosis. The report was as follows: Agglutination test with Brucella abortus, negative, opsonic test with Brucella abortus shows a Foshay index of 22; the skin test was strongly positive, the area of redness and infiltration being the size of a quarter.

On December 18th, I tried to break the adhesions in the right eye by a subconjunctival injection consisting of adrenalin 1:1,000 and atropine 2 percent. After the expiration of one hour there was no appreciable change in the pupil. The chamber was deep; tension minus. Injections of Foshay vaccine* were started at once and have been continued ever since. The dosage began at 1/20 c.c. of T-1 and has been gradually increased. At the present time the dosage is 4/20 c.c. of T-3. The injections are given three times a week. In the presence of the iris bombé and the increased tension in the left eye it seemed necessary to attempt to lower the tension by operation. Therefore on January 7, 1942, a Fuchs transfixion was performed. Despite the fact that four perforations were made in the iris and that the operative recovery was uncomplicated, the anterior chamber did not deepen, and tension remained above normal.

During the patient's stay in the Hospital the supply of Foshay vaccine gave out and could not be immediately replaced. The patient stated that since the withdrawal of the vaccine she felt "all let down" and asked that it be resumed at the earliest possible moment. January 26, 1942, it was noted that the corneal infiltrations were less dense in both eyes; there was far less tearing, and the ciliary congestion was greatly diminished.

During subsequent months a close watch was kept on the tension of each eye for it was feared that secondary glaucoma might develop in the right eye. As a matter of fact, slightly elevated tension was noted in this eye on June 15, 1942.

Tension in the right eye continued to rise slightly during June and July and reached 41 mm. Hg. (new Schiötz) on July 29, 1942. By this time the cornea had cleared remarkably so that there was an almost transparent segment between the 6- and 9-o'clock positions. Experience had proved that there was nothing to be gained by a continuation of mydriatics, and as miotics of various kinds and various strengths had failed to prevent a continuing rise in intraocular pressure it

* Hagebusch and Frei[3] state that Foshay vaccine is an oxidized Brucella abortus vaccine. This material does not cause so severe reactions as do other vaccines. The dosage has to be carefully regulated. Each patient is an individual problem as to the size of dose. Some tolerate as much as 40,000 times the amount given another patient, yet two patients with these extremes of dosage may make comparable gains in health.

seemed necessary to operate. I had in mind the creation of a new pupil corresponding to the clearest area of the cornea. I also wished to effect a freeing of the iris angle to permit the excision of the iris as near its attachment to the ciliary body as possible.

A combined cyclodialysis and iridectomy was performed in the following manner: The conjunctiva was incised 8 mm. behind the limbus. After the sclera was exposed, a slanting groove was made with the point of a keratome 6 mm. behind the limbus. This incision was 4 mm. long and ran parallel to the limbus. The incision was not completed down to the choroid. A small Graefe knife was introduced into the anterior chamber and brought out in the sclera opposite the clearest portion of the cornea. At this point the scleral cut was deepened down to the choroid, and a cyclodialysis spatula was carried into the chamber and swept through an arc somewhat less than 90 degrees. There was very little bleeding, and the iridectomy was completed without incident. The conjunctiva was sutured. At the same time it seemed desirable to supplement the first operation on the left eye, as the tension had again risen to 40 mm. Hg. (Schiötz). Accordingly, after a small conjunctival flap had been formed above, the anterior chamber was penetrated by an incision with a scalpel just behind the limbus. This incision was slightly enlarged laterally with blunt scissors. The iris was friable. Small bits were removed piecemeal.

Following the operation the patient has continued to make good progress. The cornea has continued to clear and vascularization is much diminished. There have been two brief episodes of increased tension in the right eye. The left eye has now subnormal tension. When last seen, on January 20, 1943, vision, R.E., was 10/150; L.E., light perception. Tension in the right eye was 27 mm., reduced in one hour to 20½ after the instillation of one drop of a 1½-percent solution of doryl in an aqueous solution of zephiran 1/3,500.

Summary

A young woman of rather low mentality presented herself with cloudy vision in the left eye. A well-developed iritis was found; the adhesions were so firm that they could not be broken. Subsequently deep vascular keratitis ensued. When first seen the right eye was normal, with 20/20 vision. While under observation, the right eye developed a plastic iritis, and despite prompt and energetic use of atropine the adhesions could not be broken. Subsequently, vascular keratitis occurred in the right eye. The left eye developed total posterior synechiae, with iris bombé and secondary glaucoma. The right eye, too, developed a secondary glaucoma, but without iris bombé.

While the patient was under observation at the Eye Clinic, extensive clinical and laboratory examinations were made in an effort to establish the underlying cause. Agglutination tests for Brucella abortus and Brucella melitensis were negative. This was taken as conclusive evidence of the nonexistence of brucellosis, and additional laboratory tests were not made.

On clinical grounds the condition was regarded by several consultants as tuberculous. Several courses of tuberculin, given therapeutically, failed to check the downward course of the disease. In addition to tuberculin therapy, the following measures were totally ineffective: typhoid-paratyphoid, heat therapy (Kettering hypertherm), blood transfusions, neoarsphenamine, gold therapy, exposure of the entire body to ultraviolet, sulphanilamide, thyroid, riboflavin; locally, mydriatics and miotics as indicated, short-wave

diathermy, and radium (beta radiations).

A presumptive diagnosis of brucellosis was made, based on a positive skin and opsonocytophagic test. A carefully regulated course of Foshay vaccine led to remarkable clearing of both corneas and great diminution in vascularization. Operation on the right eye—optical (basal) iridectomy and cyclodialysis—reduced the tension and greatly improved vision. Operations on the left eye (transfixion and later iridectomy) reduced tension to normal, but without visual improvement.

Conclusion

Adequate laboratory tests for brucellosis should be made routinely in any chronic inflammatory condition of the cornea, iris, ciliary body, and uveal tract. Such tests should be made early in the course of the ocular malady. If other etiology can be excluded, prompt and energetic treatment with a brucella vaccine may prove to be the means of checking the downward trend of the disease and thus preventing blindness.

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THE HEREDITARY MACULAR DEGENERATIONS

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The conditions included in this group have appeared in the literature as separate entities, with names suggested by some outstanding characteristic. The number of cases observed is not large, and because of the nature of the disease few eyes have come to the pathologist; hence our information is mostly clinical and lacks the solid ground of laboratory study without which the physician is ill at ease. Some of the types change in appearance as they progress, which adds to the confusion; there has been little correlation of case reports, and no continuous observation of family groups over a period of years. The object of this article is to interest oculists to observe family groups in which this disease may appear and to turn over afflicted eyes to pathologic laboratories of eye hospitals in the larger cities so the information we now have may be supplemented by reliable pathologic data. The names used in the literature for these various types include infantile maculocerebral degeneration and amaurotic family idiocy (Tay-Sachs); honeycomb choroiditis (Doyne); guttate choroiditis (Tay); tapetoretinal degeneration (Leber); macular degeneration (Best); progressive macular degeneration (Batten and Stargardt); retinitis punctata albescens (Gayet); and juvenile maculocerebral degeneration (Paton and Holmes). (Tay-Sachs disease is the most thoroughly studied of the group because the symptoms are so serious that the small patient is immediately placed under hospital care and the unfortunate result makes it possible to examine the tissue involved. This type is not included in this article.) Associated neuro-ophthalmologic conditions are partial or total optic atrophy, color blindness, mental degeneration, epilepsy, and pigment clumps in the periphery like those of retinitis pigmentosa. The characteristics of the group are:

Heredity and familial incidence, but isolated cases occur.

Bilaterality.

Onset—birth, second dentition, puberty, end of skeletal growth, beginning of involution, senility.

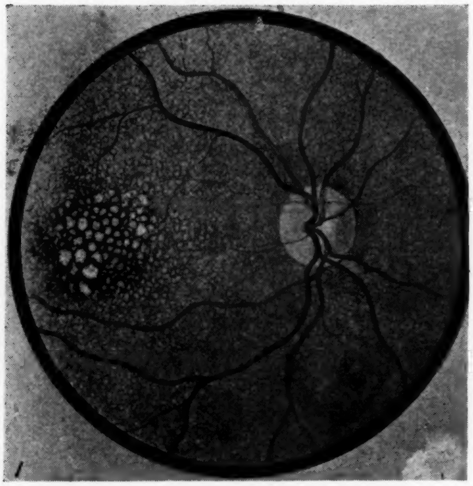
No one ophthalmoscopic picture fits all cases but the type for a family runs true.

The time and manner of onset is the same for members of a family.

If the transmitter is a female, she is usually affected, but the male transmitter may escape.

For convenience and clarity, the cases will be discussed in four groups.

The first and most common of the entire group is known by the large white spots scattered about the macular area. It appears in the literature as honeycomb or guttate choroiditis. The size of the spots varies and also the number. In some cases, the spots are very large and irregular; in other cases, they may be rather small and round, and as they spread toward the periphery, decrease in size, and merge into the fundus background, which is lighter in color than normal. In these cases, they are too numerous to be estimated. In the younger patients at least, the vision is normal (fig. 1). It is not known at what age the spots appear nor whether they change with time. The youngest patient of this type who was seen was five years old, but he has been under observation only a short time. The patients in the fifties and sixties had lowered acuity,



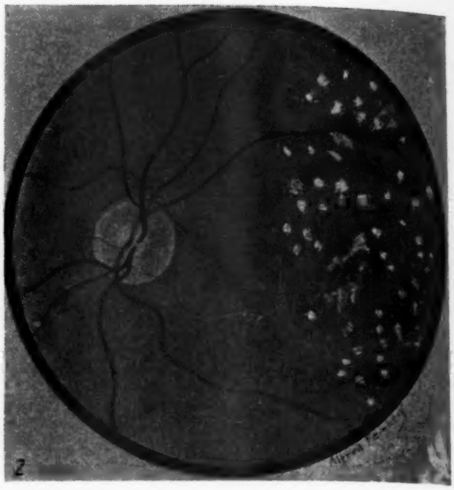
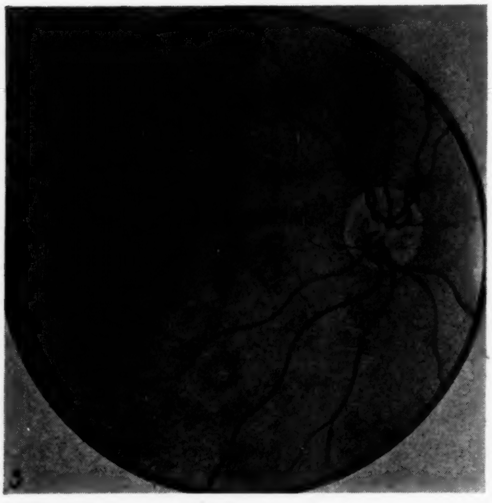


Fig. 1 (Lloyd). Type 1; vision normal at 40 years.
Fig. 2. Type 1; changes in macular region; vision 20/40 and 20/66, in a woman aged 67 years.

pigment deposits, and thinning of the choroid in the macular area (fig. 2). It is my opinion that all of these patients lose vision as they grow older but to what degree is not clear. A different type has been observed in adults of 30 and over, with round, clearly outlined spots of a red color differing from that of the fundus (fig. 3). These spots are clearly outlined and look as if rain had fallen upon dust. In the older cases, fine pigment granules were seen at the borders of the spots in the macular area. The spots were confined to the macular area in the younger patients but extended farther out toward the periphery in the older patients. It is possible that this is the earlier form of the type in which very large irregular spots are observed in and about the macular area and smaller spots farther from the fovea. The vision is normal. Another type, in which the vision is normal, gives



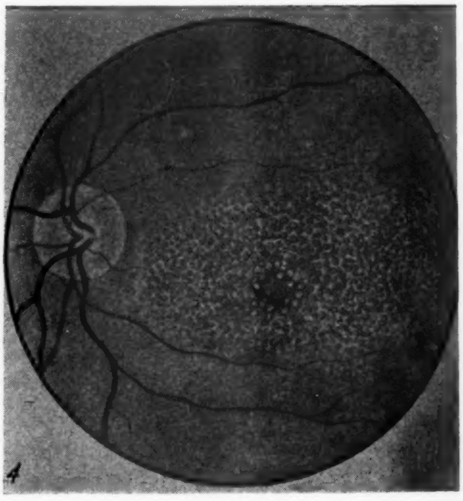


Fig. 3 (Lloyd). Type 1; aged 30 years, vision normal. "Rain drops" in macular areas.
Fig. 4. "Shad roe" found in course of a routine examination, in a woman, aged 25 years, with normal vision.

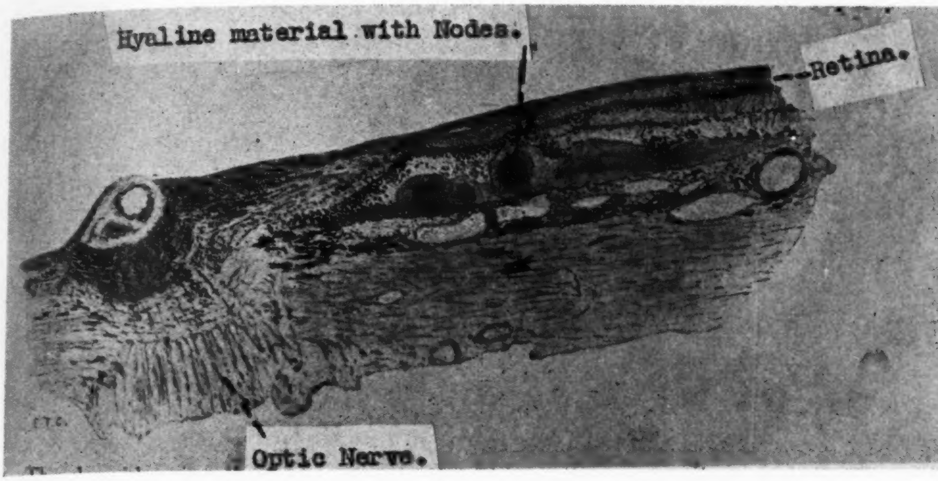


Fig. 5 (Lloyd). Pathologic report on a case of Doyne's choroiditis by Treacher Collins, in Ophthalmoscope, 1913, v. 11, p. 537.

the impression of "shad roe" in and about the macular area with thinning of the choroid between the globules (fig. 4). Only a few of this type have been observed and it may change character as the patient ages. The illustration shows the best results obtainable in black and white, but the color of the background between the globules is somewhat redder than is the unaffected area, but not on the black side as it appears here.

The lesions of all three types are so plain and numerous that it seems impossible that such extensive changes could go on without seriously affecting the vision. Treacher Collins's single report of an examination of tissue from a case of the first type follows:

Situated between the choroid and retina is a new formation of hyaline substance. It commences on each side of the optic disc close to its margin and extends inward for about two disc diameters and outwards a distance of six discs' breadth. Its outer surface has a regular contour following the natural curve of the choroid. Its inner surface presents several nodular elevations with depressions between them. The thickness varies in different parts. It is laminated in places and mostly free from cells with an occasional cell with a flattened nucleus. The choroid external to the hyaloid material showed marked thinning and in places absence of the capillary layer. The vessels of the outer choroidal layer appear larger than normal but the walls are not thickened (see fig. 5). Where the hyaline substance is present, the elastic lamina cannot be differentiated. At the periphery of this hyaline layer, on each side, the pigment layer can be seen as a single layer of cells. Large areas of the inner surface of this hyaline layer are devoid of pigment epithelium, while here and there, collections of epithelial cells remain. The outer surface of the retina is much disorganized by this tissue. In places the rounded masses are in contact with the inner nuclear layer. The granular layers are thickened in places and the fibers crossing them are stretched out with spaces between. The inner retinal layers show little change. The hyaline material looks and stains like the hyaline nodules seen in degenerative conditions of the retina. Such nodules are often termed "drusen." The hyaline material in this specimen is peculiar in forming such a long

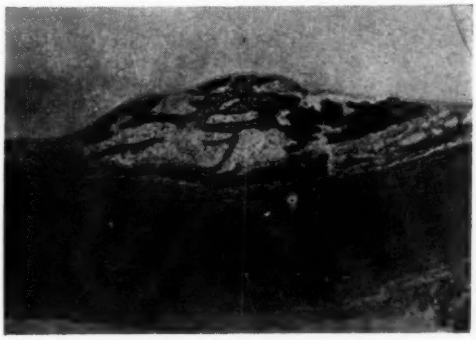
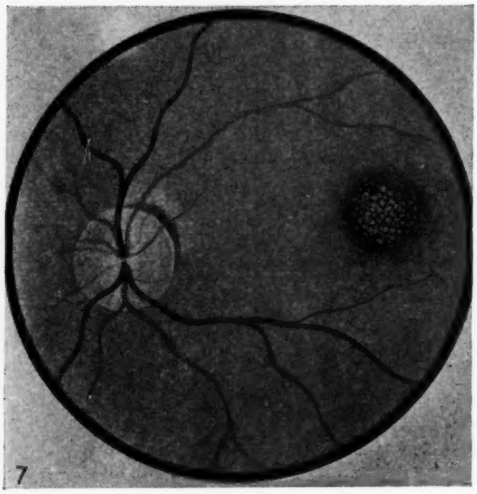


Fig. 6 (Lloyd). Drusen plaque, by E. M. Burchell.



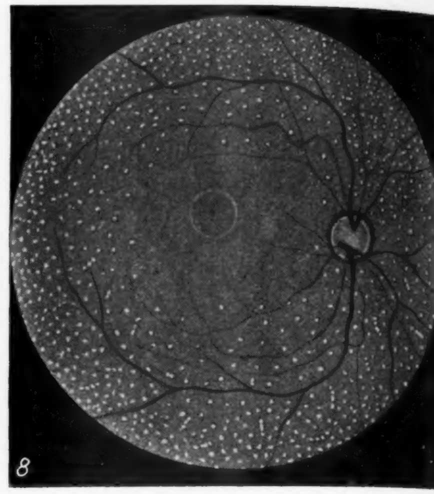


Fig. 7 (Lloyd). Senile macular degeneration. Vision is normal. The other eye has massive exudates in the macular area suggesting Coats's disease.

Fig. 8. Fundus albipunctatus cum hemeralopia congenita. By Takasu, in Klin. M. f. Augenh., 1906, v. 63.

layer. In other conditions it is in the form of isolated nodules. Different views are offered to explain this tissue. It has the same characteristics as the elastic lamina and, like it, is probably the product of the pigment epithelium. The primary change was probably in the pigment layer and the changes in the retina and choroid are probably the results of pressure.

There is no statement in this report of the age of the patient or the fundus picture.

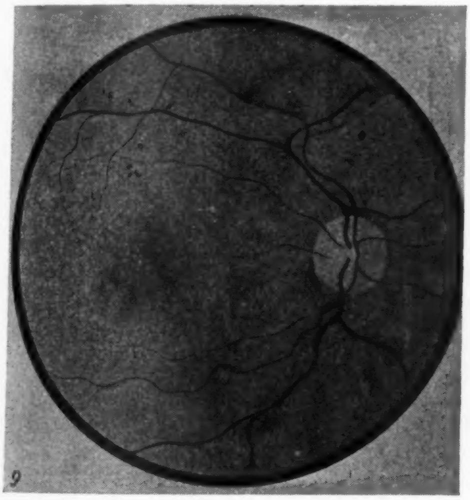
The type with large or small white spots is rather common, and cases with small white spots may be confused with retinitis punctata albescens. There is no night blindness, color blindness, nor loss of dark adaptation. In the younger patients, the normal yellow color of the macula in red-free light is retained, and the binocular ophthalmoscope locates the defects far behind the retinal vessels, evidently in the choroid. I have not had the opportunity to examine older patients who have failing vision and pigment in the macula, with red-free light. Behr considers "senile macular degeneration as a special form due to a transmitted cause producing death of the maculo-retinal neurons (see fig. 6). A simple degeneration spreads from the fovea in all directions. There is no inflammation and no pigment except what follows hemorrhage. Neither the choroid nor the nerve is affected."

Comparison with "drusen" is inevitable, but there is no reason for confusion. Ophthalmoscopically, drusen are very rare and of a different color. Microscopically, the lamina elasticum in drusen cases is very thick and has irregular extensions forward between the tissue elements. Patients with drusen are usually very old (fig. 7). Behr and others, after studying angioid stripes and cases of hereditary macular degeneration, felt that the lamina elasticum was not merely a partition but governs the transfer of nourishment to the outer retinal layers and the return of waste products to the choriocapillaris. Upon the elastic tissues of the body fall the first effects of age. The elastic layer of the arterial wall bears the chief strain, but, in the senile eye, the lamina elasticum is much thickened, with definite changes in the pigment layer of the retina. We can regard this innocent type of macular degeneration as a premature senility of the lamina elasticum, but it is impossible to imagine a disease

limited to this tissue. From every point of view, the pigment layer of the retina, the lamina of Bruch, and the choriocapillaris are a unit. All of the cases of this type were found during routine examinations for glasses.

The second type has many very small discrete white dots scattered about the fundus, most numerous in, or limited to, the periphery. The patients in whom the milder cases are found have no complaints, but careful examination reveals lowered visual acuity, reduced adaptation, and some degree of night blindness (fig. 8). Lauber suggests the name of fundus albipunctatus cum hemeralopia congenita. The early cases reported received the name of retinitis punctata albescens. This name better fits a very serious type, in which the fundus is powdered with finest white dots, and the patient is night blind, color blind, and eventually totally blind. Extended observation of these types have not been made, nor have we reports of examinations of involved tissues (fig. 9). Except the advanced type, these cases have been discovered in routine eye tests by those who would take the trouble to make extended examinations after finding the fine white dots in the periphery. From this group comes the so-called retinitis pigmentosa sine pigmento. Oguchi has reported that the white cloud of the serious form may disappear if the patient is placed in the dark for several hours, but it returns with exposure to light. There is little likelihood of mistaking this type for any other fundus lesion. Bilateral choroiditis in the early stages may show discrete white spots in the periphery, but they are much larger and fuzzy. There is no chance of confusing an early case of inherited syphilitic chorioretinitis in a child.

The third type appears in the title of juvenile maculocerebral degeneration, tapetoretinal degeneration, or progressive



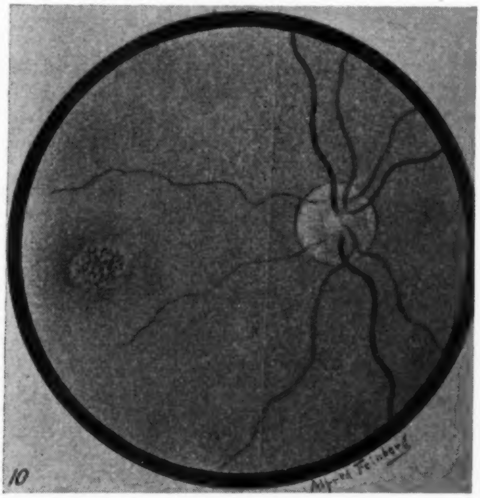


Fig. 9 (Lloyd). By Dr. Cyrus Lack. Retinitis punctata albescens. Mother and brother have normal vision, with numerous large, white spots in the maculas. Patient has been color blind, night blind, had contracted fields, but at time illustration was made had lost central vision. In making the photograph from the original illustration in color, the red background was lost, so the minute individual dots of white do not stand out clearly. Tapetoretinal atrophy is evident nasally and about the disc

Fig. 10. Juvenile macular degeneration. Onset at 10 years of age; vision two years later was 20/70. Two sisters in one family, but no other cases found.

macular degeneration, according to the stage in which it was recognized (fig. 10). These cases come to examination early because of poor vision. A fine pigment is seen in the macular area, with thinning of the choroid. There is a central scotoma, but the macular changes may not appear until a year or more after the patient finds his vision is fading. A ring scotoma develops, the peripheral fields contract, and along with this goes an atrophy of the tapetum, usually first

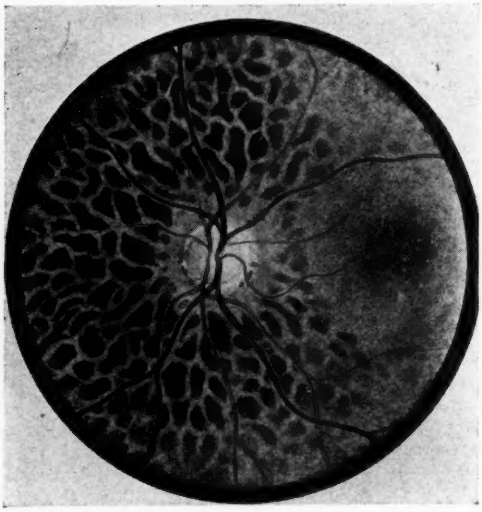


Fig. 11 (Lloyd). Advanced form of type 3. Ring scotoma, reduced vision, contracted fields, night blindness. Onset at 20 years of age. The advanced thinning of the tapetum and sclerosis of the choroidal vessels are evident. First complaint: poor vision.

in the nasal periphery, disclosing the vessels of the choroid to be affected with beginning sclerosis. The ring scotoma breaks through at the horizontal meridian, the upper and lower functioning fingers shrink, and the patient becomes blind. Color blindness may be an early sign, and dark adaptation suffers eventually, but sometimes early. The optic nerve atrophies, but the pallor may not appear until late. If the symptoms appear before puberty, the patient usually degenerates both physically and mentally and dies an imbecile. The onset, progress, and details of the cases vary with the family, but the type is usually the same for each group (figs. 11 and 12). From these cases have come tissues which show that the condition is very similar to the infantile form of maculocerebral degeneration. Paton and Holmes studied three cases in one family with the age of incidence at 10 years. They report:

> There was loss of vision, tapeto-retinal degeneration, atrophy of the disc, and shrivelled vessels. The microscope revealed degeneration of the outer retinal layers and replacement with proliferated neuroglia. The inner retinal layers (nerve fibers, ganglion cells, inner reticular, and nuclear layers) were little affected. The earliest change was a breaking up of rods and cones, and rarefaction and thinning of the outer nuclear layer. There was also an atrophy of the cerebellar cortex, disappearance of its granular layer, diminution in number of Purkinje cells, and the fiber network about them.

There seems to be no other fundus lesion that would cause confusion in diagnosis. Isolated macular lesions of tuberculous origin in young people occur but are not confined to the macular area on both sides. A bilateral macular lesion of specific origin, characterized by heavy black pigment in the choroid and secondary involvement of the retina, occurs in adults but absence of a ring scotoma, night blindness, and the progress of the case would distinguish it.

The initial lesions of the last type are macular and appear as if some of the layers of the choroid were missing. The edges of the defects are sharp, the sclera is not bared, but long, straight choroidal vessels may be uncovered. This might be said to resemble a partial coloboma of the choroid (fig. 13). Ordinarily, there is little pigment in or about the lesion, but in some cases there is a vicious pigment reaction, with clumps of heavy pigment.

In this type the peripheral choroid may resemble gyrate atrophy of the choroid and retina, or the type of choroideremia which is really retinitis pigmentosa with an overgrowth of neuroglia.

None of the cases of this group have been watched through the various stages, but patients who were thought to be in the early stages showed no loss of vision, even those with a considerable defect. It is impossible to say from the ophthalmoscopic appearance what the vision will be, for the two fundi of a patient may look alike but the visual acuity be 20/20 in one eye and 20/50 or worse in the other. One of these cases concerned an elderly lady in her sixties who had been able to read the paper until just before her visit (fig. 14). Vision with her glasses was 20/100 and she could not read type even much larger than newsprint. The presbyopic correction in use was +4.00D. sph., which leads to the conclusion that, like other macular lesions, function is re-

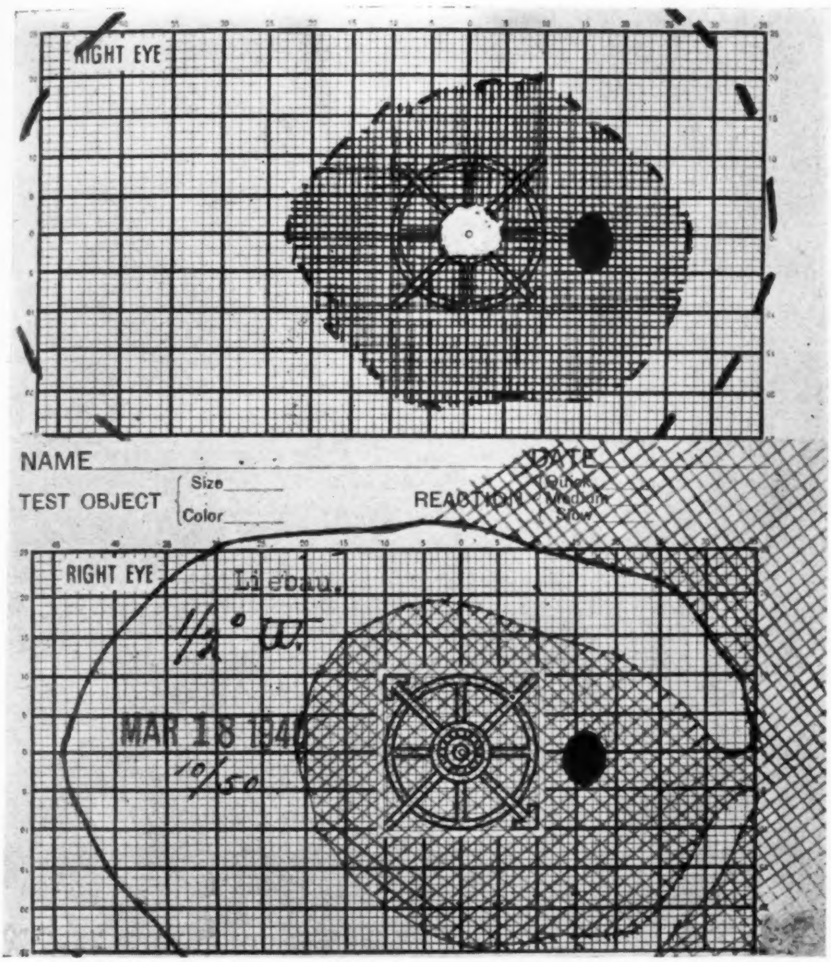
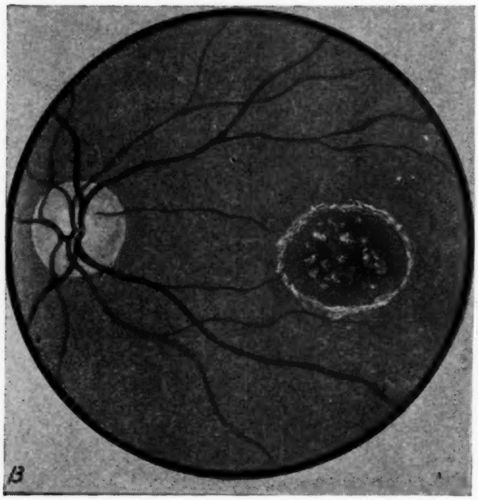


Fig. 12 (Lloyd). Ring scotoma from preceding case. The charts were made seven years apart, and the breaking through of the ring defect is seen in the horizontal meridian in the later record.



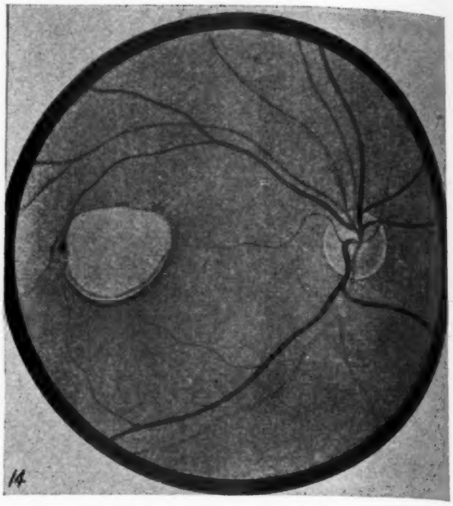


Fig. 13 (Lloyd). Early stage of the fourth type of macular degeneration with normal vision.

Fig. 14. Woman of 60 years had sudden failure of vision to 10/200. Had been able to read the paper and do her work without difficulty.

tained for a long time but finally shows a limited viability. This lesion seems to be an absorption and atrophy of the choroid in the macular region with later involvement of the retina. This type must be differentiated from extrapapillary coloboma; a stationary congenital defect in which there is pigment at the edges or within the defect, but no color blindness or other indication of progressive disease of the pigment layer of the retina. The amount of pigment in both diseases varies amazingly. Macular degeneration never has the sclera exposed to view, with ectasia, as is seen in coloboma. Vascular communications of the choroidal and retinal systems may be found in the coloboma but not in macular disease. The milder colobomas may be confused with the early stages of macular degeneration, but a period of observation will make the diagnosis certain. While the coloboma patient may not be able to read the newspaper, if the lesion is bilateral, the peripheral retina functions well and the patient is able to go about the streets with

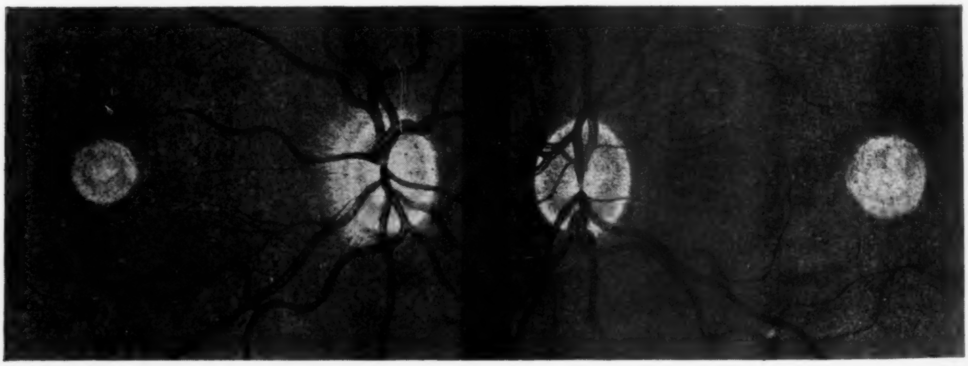


Fig. 15 (Lloyd). From Adams: Transactions of the Ophthalmological Society of the United Kingdom, 1883, v. 3, p. 113. Type of congenital coloboma which might be confused with type 4. The amount of pigment varies in both conditions.

little trouble (fig. 15).

In cases of this fourth type, with pigment reaction, there are all the symptoms that go with pigmentary degeneration of the retina. In addition, the periphery may be involved by what appears to be choroidal atrophy, suggesting gyrate atrophy of the choroid. In neither gyrate atrophy nor pigmentary degeneration are there macular lesions. The secondary cataract so characteristic of pigmentary degeneration is not found in cases of macular degeneration. In the early stages, the macula retains its normal yellow color when viewed with red-free light, and only the bolder choroidal lesions are seen. The yellow color of the macula disappears as the disease advances, and soon there are many opaque dots, spots, and lines in the retina itself.

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COMPARISON OF THE KERATOME–SCISSORS AND GRAEFE-KNIFE INCISIONS FOR CATARACT EXTRACTION*

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Three years ago, in an effort to simplify and improve cataract operations, we began making the incision with an angular keratome and scissors instead of with the Graefe knife. This type of incision proved so simple and successful that now it is invariably used. As time went on and a large number of cases accumulated, it was noted that, after the keratome–scissors incision, the postoperative astigmatic error was of unusually low degree and there seemed to be a general improvement in the average acuity of vision. This surmise is borne out by statistics compiled from 220 consecutive cases, in 110 of which the incision was made by the keratome–scissors technique and in 110 with the Graefe knife. These cases were not selected but were reviewed in chronologic order and included operations by all members of the teaching staff, along with the younger and older members of the interne staff.

From personal experiences as the surgeon or assistant in more than 3,000 extractions made with the Graefe knife and probably 700 or 800 with the keratome–scissors, it is our impression that the latter method is the preferable one, a belief based principally on observations in the teaching of surgery.

There can be no doubt that the keratome–scissors incision is better for beginners and for those who operate infrequently. The operation is not approached with fear and trembling such as that often observed in those about to make an incision with the Graefe knife. The keratome is easier to handle, and it is less difficult to place the incision in its proper location. There is no counterpuncture which may be too deep or too shallow. The keratome incision need not be rapid but is made slowly and carefully, without incurring loss of aqueous and with time for any necessary change in direction as it is continued. Really, it is difficult to make a poor keratome incision. Furthermore, fixation need not be so secure, since the keratome assists in steadying the globe; also, if fixation is lost, it is easily regained. In addition, there is no liability to rotation of the eyeball, as often occurs during the stages of puncture and counterpuncture with the Graefe knife. There can be no question but that

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the keratome–scissors incision is smoother and therefore heals more kindly; this has been confirmed by microscopic study of eyes removed several months or years after cataract extraction. There is little bleeding from the keratome–scissors incision, since the vessel walls undergo some compression and are not cut so cleanly as with the Graefe knife. Because of its broader surfaces the wound heals rapidly, the anterior chamber soon reforms, and wound separation in less frequent. This type of healing is attended with less corneal astigmatism than is the more ragged Graefe-knife incision; also the astigmatic error is less likely to be at an oblique axis, since the incision may be more accurately placed and controlled.

The incision is made with a wide-angle keratome, and its point is inserted two or three millimeters behind the upper limbus at the 12-o'clock position; the knife is carried downward through the anterior chamber, just in front of the iris, until the point almost enters the cornea below. After withdrawal of the keratome the incision is carried downward to the horizontal meridian on either side with a small, blunt-pointed, curved scissors. Because of its slanting nature and a tendency to be smaller than it appears, the incision should be made slightly larger than the usual Graefe-knife incision; that is, to the horizontal meridian. Sutures are easily placed in the episclera and limbic tissues after such an incision. One should never make the iridectomy until the incision has been completed with the scissors—if it is made after the keratome is withdrawn and before enlargement of the incision with scissors, the iridectomy is more difficult and the iris and lens capsule are liable to injury.

Apparently the keratome–scissors incision does not reduce the number of fourth- to sixth-day anterior-chamber hemorrhages, although one might expect it to do so since the vessels are more or less pinched off during the incision.

That there is less astigmatism following the keratome–scissors incision cannot be doubted after study of the following statistics (table 1). All operations were attempted intracapsular extractions, and the results listed below were based on refractions by various members of the staff,

TABLE 1

ASTIGMATISM AFTER CATARACT EXTRACTION (220 CONSECUTIVE CASES)

Astigmatism (in diopters)	Keratome–Scissors Incision—110 cases	Graefe-Knife Incision—110 cases
1.00 or less	41 cases	13 cases
1.50 or less	63 cases	30 cases
1.25 to 2.00	35 cases	35 cases
2.25 to 3.00	25 cases	41 cases
3.25 to 4.00	5 cases	10 cases
4.25 to 6.00 (k*) 7.00 (g*)	2 cases	10 cases
Undetermined†	2 cases	1 case
Average astigmatism	1.64 diopters	2.42 diopters

(k*) keratome incision; (g*) Graefe-knife incision.

† One case of postoperative iris prolapse and irregular astigmatism (6/30 vision). One of vitreous loss with loop extraction (6/60 vision). One of preoperative and postoperative iridocyclitis and glaucoma (vision −6/60).

two months after operation. It is realized that astigmatic errors often decrease with the passage of time and many of these errors probably lessened. All types of patients were included—good, bad, and indifferent; also all types of senile cataract—that is, incipient, intumescent, mature, and hypermature; also those with a good or bad preoperative prognosis. Included also were all cases in which there was such involvement as postoperative anterior-chamber hemorrhages, iris prolapse, wound separation, iridocyclitis, optic neuritis, and glaucoma.

Central visual acuity following the two types of incision did not differ greatly; however, a larger number of patients with exceedingly acute vision had been oper-

ated on by the keratome–scissors technique (table 2). Fourteen patients—singularly enough, seven in each series—were given a poor prognosis before operation. Other statics of interest are as follows:

1. Each of the 220 operations was an attempted intracapsular extraction. The lens was removed by this method in 168 operations (76 percent).

2. Needling was necessary in 4 of the 52 extracapsular cases (7.7 percent).

3. The only recorded accident at operation was loss of vitreous in two cases (slightly less than 1 percent).

4. Postoperative complications of sufficient severity to affect vision were (a) iris prolapse in one case, (b) wound separation in two cases, (c) iridocyclitis in three cases, in one of which also an optic neuritis developed such as one sees occasionally after cataract extraction.

5. Other causes of poor vision, recognized in 10 cases before operation, were: corneal scarring, pterygium, vitreous in the anterior chamber, chronic iridocyclitis, progressive myopia, diabetes, and glaucoma simplex.

6. After operation, in five cases, poor vision appeared to be due to macular degeneration or optic atrophy.

TABLE 2

VISION AFTER CATARACT EXTRACTION (220 CONSECUTIVE CASES)

Vision	Keratome–Scissors Incision 110 cases		Graefe-Knife Incision 110 cases	
	Intracapsular	Extracapsular	Intracapsular	Extracapsular
Better than 6.6	15	3	10	0
6.6 or better	57	15	66	7
Approximately 6.9	10	9	14	8
Approximately 6.12	4 (2*)	5 (2*)	1	0
Approximately 6.15	2 (1*)	1 (1*)	4	2 (1*)
Approximately 6.21	0	0	5 (4*)	2 (2*)
Approximately 6.30	2	0	0	0
Approximately 6.60	2	1	1 (1*)	0
Light perception	1	1 (1*)	0	0

* Poor preoperative prognosis (see 5, in the following statistics).

SUMMARY AND CONCLUSIONS

1. The keratome–scissors incision for cataract extraction is simpler and safer than the Graefe-knife incision.

2. Astigmatism is less after the keratome–scissors incision. In 110 consecutive extractions through keratome–scissor incisions, the astigmatism averaged 1.64 diopters, whereas, with a like number of Graefe-knife incisions, the astigmatism averaged 2.42 diopters.

3. There was little difference in acuity of vision aside from the fact that a greater percentage of those with very high acuity were operated upon with the keratome–scissors technique.

GLIOMA OF THE RETINA IN SUCCESSIVE GENERATIONS

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Although the tumors known as glioma retinae, or retinoblastoma, usually manifest themselves in apparently isolated or sporadic cases, instances of the familial occurrence of these tumors have been noted for many years. A case often cited is that reported by Newton[1] (1902), who observed a family of 16 children, 10 of whom had retinal glioma. Wilson[2] (1827) reported eight childen in one family, all of whom were afflicted with glioma retinae. Von Lerche[3] (1821) reported a family of seven children in which three boys and one girl had retinal glioma. Sichel[4] mentioned three cases of glioma in one family. Von Graefe[5] (1868) referred to the discovery of glioma in two of a family of six or seven children. Calderini[6] (1867) mentioned 3 daughters afflicted with retinal glioma in a family of 14 children. Schönemann[7] (1880) saw two cases of glioma in a family of three children. Macgregor[8] (1885) reported three cases of glioma of the retina in a family of five children. Flexner[9] (1891) observed that retinal glioma of the type which he called neuro-epithelioma affected three children of one family. Brown[10] (1891) reported three cases of glioma retinae in one family.

Marshall[11] (1897) reported a family of six children, three of whom had tumors of the eye. Two of the children were dead; one was living 14 years after enucleation of the affected eye. Boyd[12] (1898), in discussing a paper by Thompson,[13] mentioned a patient who died of glioma retinae. This patient was one of a family of five children, and a brother and sister had died of the same cause. Cirincione[14] (1902) and Calderaro[15] reported that four of five children in a family had glioma. Schönemann (1880) and Valenti[16] (1903) each saw instances in which two of three children were affected. Snell[17] (1904) reported two families, in each of which a brother and sister had glioma. In one family the tumors were bilateral in both cases. Adam[18] (1911) saw three families, in each of which two children had glioma retinae, a brother and sister in one family, two brothers in each of the other two families. Dabney[19] (1915) reported that three children of one family died of retinal glioma. The lesion was bilateral in two of the cases but involved only one eye in the third case. Leber[20] (1916) mentioned a family of nine children, three of whom had bilateral retinal glioma. Comas[21] (1920) reported a family in which 5 of 11 brothers and sisters had glioma of the retina. Fuchs[22] (1933) reported that two of three children had glioma. R. Foster Moore[23] (1937) saw two brothers, each with bilateral glioma. Weller[24] (1941) observed 6 children with glioma in a family of 10 children; 4 of the children had bilateral tumors. Falls[25] (1942) discussed the same family mentioned by Weller (1941).

An interesting case was reported by Townsend[26] in 1939. A Negro woman had a daughter who had glioma retinae. The mother later bore two daughters by another father, both of whom had glioma. There was no record of a tumor of the eye in the mother or either of the fathers.

Although such instances of the familial occurrence of retinal glioma have been recorded for many years, reports of the observation of glioma in succeeding gen-

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* From the Section on Ophthalmology and the Section on Surgical Pathology of the Mayo Clinic.

erations have been rare until recent years. Parsons[27] questioned whether proof of such an occurrence had ever been established. Von Hoffmann,[28] who in 1908 reported the case of a woman who had had a gliomatous eye removed at the age of two years, and who, 28 years later, had had a child who had a glioma in its first year of life, claimed that there was no other such case on record. Of recent years additional reports of glioma occurring in succeeding generations have come to light, and Weller (1941) collected 30 cases, 29 reported by other authors and 1 reported by himself, in which retinoblastoma has affected more than one member of a family, other than siblings alone. These cases included not only those in which there apparently was direct transmission from parent to child, but also those in which the disease occurred in collateral branches of a family through nondemonstrating parents. He also included the case reported by Townsend, in which half-sisters were affected with glioma retinae. The case reported by von Hoffmann in 1908 has already been mentioned. In 1916 Leber reported a communication from von Hoffmann, which furnished further information on this case. The first child had bilateral glioma but only the right eye was enucleated. A second child, a boy, had bilateral glioma at the age of four months. Both these children died. A third child was well at seven months of age. Despite von Hoffmann's claim that he reported the first such case, it would appear that several fairly well-authenticated reports of glioma occurring in succeeding generations had appeared previous to his report. In 1910 de Gouvea[29] republished a report originally made by him in Portuguese, in 1886, of a case in which a man had had his right eye removed for glioma at the age of two years. Two of his seven children died of bilateral retinal gliomas. This is apparently one of the first reports of a case in which glioma affected a parent and child. Johnson Taylor[30] (1905), in discussing a paper by Snell,[31] briefly mentioned removing the gliomatous eye of a child whose mother had had an eye enucleated in early life, presumably for glioma. Owen[32] (1905) reported the case of a man who at the age of five months had had his left eye removed for glioma, and whose son 39 years later had his right eye removed at the age of three years for glioma. The sister of the last-mentioned patient, whose eyes were normal, had two children who also had glioma of the retina. Diagnosis of glioma in the case of the son was confirmed by microscopic examination.

Berrisford[33] in 1916 gave a further account of the family reported by Owen. At that time four of eight children of the sister had glioma (three had bilateral glioma). Lukens[34] (1908) is said to have reported a case of glioma in a patient who had a cousin who died of the same disease.[35]

. Caspar[36] enucleated the left eye of a girl, two years of age, for glioma of the retina; 19 years later, in 1911, the daughter of this patient had her right eye enucleated for a retinal glioma which filled the globe.

De Haas[37] (1916) (quoted by Weller) reported the case of a child who had bilateral retinal glioma and had the right eye enucleated in 1913. The father of the child had had his right eye removed for glioma in 1882.

Griffith[38] (1917) reported two families with glioma in two generations. In the first, two brothers and two sisters of a family of six children had bilateral glioma retinae. The mother, at the age of nine months, had had her right eye removed for a growth reported to be exactly like that of one of her sons. The second family included three children, two

sisters and one brother, each of whom had undergone enucleation for glioma (bilateral in one case). Their mother, at the age of two-and-a-half years, had had her right eye removed because of glioma. According to Griffith, these cases were all typical examples of the disease, but no mention was made of a microscopic examination of the tumors.

Pockley[39] (1919) reported a family in which one of the members had been operated on earlier by Maher[40] (1902). A father who had had both eyes removed by Maher for glioma, one at the age of 18 months, the other at the age of two years, had a child who, at the age of 18 months, lost an eye because of glioma. The diagnosis in the case of both the father and the child was confirmed by microscopic examination. There was also a horizontal familial incidence in this case; two sisters of the father had had both eyes removed for glioma in infancy.

Traquair[41] (1919) gave the details of a family in which the father, at the age of six months, had lost his left eye on account of glioma. The first child was stillborn, the second child, a son, and the third child, a daughter, each had glioma retinae. The disease was bilateral in the case of the son and involved the left eye in the case of the daughter. Fietta[42] (1925) observed a case in which the mother had lost her right eye at the age of 19 months because of glioma. Her daughter also had glioma of the right eye, which was enucleated when she was 22 months of age. Van der Hoeve[43] (1926) reported a family in which the father, who had had one eye removed for glioma at the age of three years, had 12 children, two of whom had bilateral glioma.

Odinzow,[44] in a footnote to a paper by Sabugin[45] (1927), mentioned a case of glioma in which the patient was the daughter of a woman who, as a child, had lost one eye because of glioma. Sym[46] (1928) reported a case in which a father, who had had his left eye removed at the age of nine months because of glioma of the retina, had two children who died of bilateral glioma when they were approximately four years of age. In the same family there was one stillbirth and one child who was reported well at the age of three years. Letchworth[47] (1928) reported a family of five children, one of whom was stillborn. One was living and well, but three boys had glioma retinae. The father had lost his right eye, at the age of two years and three months, because of glioma. Griffith[48] (1933) and Hine[49] (1937) gave further reports on the same family reported by Letchworth. Two of the sons, who had bilateral glioma, were dead; one son, who had had his right eye removed because of glioma at the age of three months, was living and well 15 years later. Definite mention was made of microscopic examination of the tumor in the case of one of the sons.

Meyer-Riemsloh[50] (1929) reported a case in which a boy had had his left eye removed at the age of six months for glioma. Diagnosis was confirmed by microscopic examination. His father, when three years of age, had lost his right eye because of glioma.

Best [51] (1934) reported the occurrence of bilateral glioma in two of three children, one of whose parents had glioma of one eye. Stallard[52] (1936) mentioned a family in which the father had bilateral glioma retinae and two sons also had bilateral glioma. In the case of one of the sons, the diagnosis was made at the age of two-and-a-half years; in the case of the other son the diagnosis was made soon after birth. It is interesting that, although the father had bilateral glioma, only the left eye had been enucleated (at the age of 14 months); microscopic examination of this eye had confirmed the diagnosis of glioma. Operation on the right eye

was refused, but at the age of four years the patient had a severe attack of scarlet fever and "the eye cleared." In 1934, when the patient was 34 years old, there were two irregular pale areas of retinal atrophy with pigmentary disturbance near the equator and the nasal branches of the central retinal vessels, but there was no evidence of neoplasm and no defect in visual acuity. Reiser[53] (1937) reported the case of a mother who had lost her left eye, at the age of one year, because of glioma. Her son died of metastatic involvement after removal of his left eye for retinoblastoma.

In 1938 Lange[54] published a report of two families with glioma. In one, the father, at the age of four years, had had his left eye enucleated for a tumor which upon histologic examination showed a typical glioma with formation of rosettes. Two sons had bilateral glioma retinae. The diagnosis in both instances was confirmed by histologic examination, which showed typical glioma retinae with rosettes. This is one of the few reports in which microscopic diagnosis of the tumor in two generations was mentioned. In the other family reported by Lange, the father had had one eye removed for tumor at the age of one year and nine months; his son lost his left eye at the age of one year and nine months because of glioma and the next year his right eye was found to contain a glioma.

Weller (1941) reported a family in which the father had had his left eye removed at the age of two-and-a-half years because of glioma, which was diagnosed grossly and microscopically. Two of the daughters had glioma; in one case the disease was bilateral and in the other it involved the left eye. Microscopic examination of the tumors in the children was not specifically mentioned, but it was stated that the "diagnosis was verified." This family is interesting in that there was a history that the father's father had had one eye enucleated at the age of three years, and that his father had had one eye removed in childhood. As Weller pointed out, if it may be assumed that the eyes of the grandfather and great-grandfather were removed for glioma, this is a unique instance of glioma occurring in four successive generations.

In addition to the instances of glioma occurring in successive generations, there are several reports of cases in which there appears to have been indirect transmission of glioma through a "nondemonstrating" parent. One of the earliest of these reports was made by von Graefe (1868). The patient was a child whose parents had normal eyes but whose mother had several siblings with "eye cancer."

Thompson and Knapp[55] (1874) made an interesting report of a family in which the parents were free from disease of the eye but a son and daughter each had glioma of the retina, and a cousin on the father's side died of glioma of the left eye. There was also a history that the father's aunt had lost two children, between their second and fourth years, because of the same disease. Thompson in 1898 reported further on this same family. At that time, 5 of 14 children (2 boys and 3 girls) had died of glioma. Thompson said that the father's great-aunt was reported to have had three children who died of cancer of the eye.

Steinhaus[56] (1900) reported a family of 12 children, 3 of whom had unilateral glioma of the retina. One of the children who did not have glioma (the first-born son) became the father of two children, a boy and a girl, each of whom had the left eye removed for glioma.

Purtscher[57] (1915) gave the history of a family of 11 children, of which 2 boys died of bilateral glioma retinae. A sister, with normal eyes, had a son who died of glioma of the left eye. In addition, another sister had abnormal eyegrounds and a nearly blind left eye. It was stated

that this may have represented the spontaneous regression of a glioma.

Sabugin (1927) observed two brothers who had bilateral glioma, one, two years of age; one, eight months of age. He reported that the mother's brother also had retinal glioma, of which he died.

Waardenburg[58] (1932) is said to have quoted Hemmes[59] (1931) as reporting an instance in which a normal father who had four siblings affected by glioma retinae, had four children with retinoblastoma. He also was quoted as reporting a case of bilateral glioma in which the patient was a girl whose father had had one eye removed because of glioma. However, we are unable to find any report of such cases in the original article. Hemmes stated that he found no evidence of a hereditary tendency in cases of retinal glioma; in the 48 cases which he reported, none of the parents of the patients had had an eye removed in childhood, none of the siblings or their children had had eyes removed or had died of ocular disease, and none of the nine children later born to four gliomatous patients had glioma.

Report of cases

We wish to report four cases which illustrate the hereditary occurrence of glioma of the retina. In all of these cases the clinical diagnosis was confirmed by microscopic examination.

Case 1. A girl, aged 11 months, was referred to the Mayo Clinic on August 9, 1937, because of bilateral glioma of the retina. Four days previously, the mother first had noticed that the child's left eye was red. Although the child had cried and had held her hands to her head, she had not been ill. The mother had thought that these symptoms were due to pain in the region of the left eye. The child was taken to an ophthalmologist, who made a diagnosis of bilateral glioma of the retina and referred the patient to the Clinic. The mother had had a similar tumor in her own left eye at the age of seven months and the eye had been removed. Her right eye had remained unaffected. The father was well and his eyes were normal.

Examination of the child's right eye revealed two confluent tumorlike masses extending into the vitreous on the nasal side. In the left eye there was considerable detachment of the retina behind which was a semitranslucent mass that threw the retina into folds and lobes. A roentgenogram disclosed calcification in the left orbit.

The left eye was enucleated on August 12, 1937. The optic nerve near the globe seemed normal in appearance; therefore, the orbital portion of the nerve was not excised. Microscopic examination of the optic nerve revealed no neoplastic cells.

The right eye was treated with radium from August, 1937, until the end of February, 1940. The mass in the eye seemed to shrink and then to grow again. The child remained fairly comfortable until early in 1940, when it became quite evident that the tumor was growing in spite of the treatment with radium. On January 26, 1939, radium needles had been inserted into the tumor, and in May, 1939, radium needles again had been implanted in the tumor of the right eye. However, the growth of the tumor was not checked and the eyeball was removed elsewhere in 1940. Sections from the enucleated right eye were sent to us for examination. Microscopic examination revealed that the tumor had the typical structure of glioma; that is, it was composed of perivascular bands or cuffs of tumor cells, surrounded by a mantle of degenerating cells. The tumor cells were small, with scanty cytoplasm and nearly spherical or polygonal hyperchromatic nuclei. Many of the tumor cells were grouped in irregular rings about clear spaces containing fibrillar material, forming pseudorosettes. In some regions the tumor seemed to be arising in the ganglion-cell layer of the retina; in other regions the tumor was diffuse and no point of origin could be made out. No invasion of the optic nerve was found. The tumor was classified as retinal glioma of neuroepithelioma type.

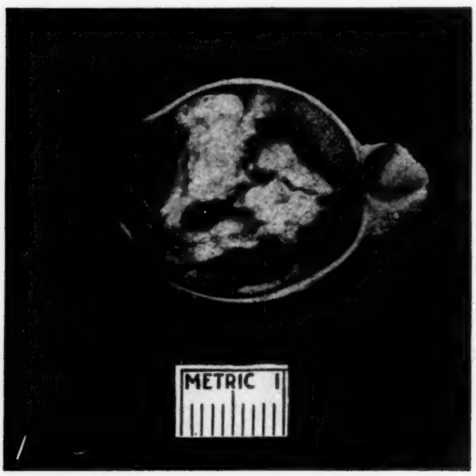
Case 2. A girl, aged four years, who was the twin sister of the patient in case 3, was brought to the Clinic November 30, 1922. In the latter part of February, 1922, her parents first had noticed a yellowish-white reflex through the pupil of her left eye. In the next few months, her left eye occasionally had become very painful and inflamed.

External examination of the right eye revealed no abnormality. Ophthalmoscopic examination of this eye, which was made while the pupil was dilated, did not disclose any lesion of the fundus or of the media. Examination of the left eye revealed slight pericorneal injection of the conjunctiva. The cornea was clear but the anterior chamber was very shallow. Only the rim of the iris could be seen at the limbus. The pupillary region was filled with a light brown mass. The lens was opaque and the fundus could not be seen. On trans-

illumination the light rays were poorly transmitted. A diagnosis of retinoblastoma was made, and the eye was enucleated on December 7, 1922.

The eyeball was enlarged and the sclera was very thin. The optic nerve apparently was filled with tumor tissue for a distance of three quarters of an inch (1.91 cm.) beyond the eyeball (fig. 1). Beyond the point of involvement, the size of the nerve appeared to be normal. There was a distinct line of demarcation in the course of the nerve, which apparently marked the extent of the glioma. The vitreous chamber was filled with a large, opaque, creamy white mass that was soft and fungating. There had been extensive invasion of the choroid posteriorly. In this region, the tumor appeared firm and white, although it was stained brown in certain regions. Microscopic examination revealed that the thickened portion of the optic nerve was densely infiltrated with tumor cells which had undergone extensive necrosis. Examination of the tumor within the globe revealed perivascular bands of tumor cells that were surrounded by extensive regions of degeneration. No rosettes were observed. Although the cells manifested some tendency to be arranged in the form of pseudorosettes in the choroidal extension of the tumor, the tumor consisted predominantly of cells of extremely undifferentiated type; therefore, it was classified as a retinoblastoma type of glioma.

The socket healed without complications. On February 9, 1923, an artificial eye was fitted and the patient was dismissed. On March 19, 1923, the patient returned to the Clinic because of pain which had been present over the left orbit for the previous three weeks. She had vomited several times daily and there had been an increase in her temperature. There was evidence of considerable emaciation. Inspection revealed slight fullness of the left orbit but palpation did not disclose a definite mass. Examination of the right eye did not disclose any abnormality. On March 30th, an exploratory operation was performed on the left orbit and a specimen was removed for microscopic examination, which revealed a glioma of the same nature as that found previously in the left eye. The posterior half of the orbit was filled with a semisolid mass, which presumably was a recurrence of the original tumor.



Figs. 1 and 2 (Benedict and Parkhill). Glioma of the retina. Fig. 1, gross appearance of tumor of left eye in case 2; the optic nerve is greatly enlarged. Fig. 2, gross appearance of tumor of the left eye in case 3; the tumor involved chiefly the nasal and posterior portions of the retina.

The patient was permitted to return home on April 9th. The prognosis was considered poor. She returned to the Clinic on May 3d, and died of cerebral extension of the growth on May 26, 1923.

Case 3. The patient in this case was the twin sister of the patient in case 2. The two girls were identical in appearance and were brought to the Clinic at the same time. In the latter part of August, 1922, the parents of the patient first had noticed a white growth in the lower nasal quadrant of her left eyeball. The size of the growth apparently had increased gradually until the vision of the affected eye practically had been obscured.

The size, shape, and position of the eyes were normal. The lids opened and closed properly. The conjunctiva and cornea of both eyes were clear. The anterior chambers were normal, and the pupils were equal and reacted normally to accommodation and to direct and consensual light.

Ophthalmoscopic examination of the right eye revealed that the media were clear and that the optic disc was round and of good color. In the lower temporal quadrant of this eye there was a detachment of the retina. The retina was elevated about 5 diopters. Many round, grayish-white spots were visible. There was a deposit of pigment at the margin of the detached retina. In the central part of the detached retina, a portion of the retina, which was situated at about axis 30 and in an oblique position, had an irregular contour and was of a lighter red color than the rest of the retina. The central portion of the detached retina had a dense, grayish-white appearance. A diagnosis of glioma of the right eye was made.

An elevated white mass, about 7 mm. in diameter, was visible in the lower nasal quadrant of the left eye. This mass was situated behind the iris. Retinal vessels were visible over the mass, which was irregular in outline and occupied a sector of the retina corresponding to that situated between the figures 7 and 9 on the dial of a clock. There was a marked alteration of the retina and choroid about the mass. Many large and small opacities were present in the vitreous. The pupil of this eye dilated widely and freely. A diagnosis of glioma of the left eye also was made.

The left eye was removed on December 7, 1922. The optic nerve beyond the eyeball appeared to be normal (fig. 2). No growth could be felt in the orbit after the eye had been removed. The socket was in good condition when the patient was dismissed from the hospital. The retina on the nasal side had been destroyed almost completely by the tumor, which extended over the temporal side of the retina posteriorly. Microscopic examination revealed the usual arrangement of tumor cells into perivascular bands (fig. 3). True rosettes were not observed but pseudorosette formation was extensive. A few tumor cells were present in the optic disc but there was no evidence that the tumor had invaded the optic nerve. The tumor was classified as a neuro-epithelioma type of retinal glioma. It was similar to the tumor encountered in case 4.

Radium was applied to the right eye in December, 1922; in February, 1923; in April, 1923; in September, 1923; in December, 1923; in April, 1924; in October, 1924; and in April, 1927. The dose at each treatment was 6,000 milligram hours. The tumor receded in size, grew again, and then receded. By May 9, 1928, the original tumor had disappeared almost completely, but a posterior cortical cataract had developed and reduced the vision. A preliminary iridectomy was performed on October 4, 1929. On November 4, 1929, the cataract was removed by linear extraction. In the course of the operation, a considerable amount of black fluid, which probably was degenerated blood, escaped from the eye. There was a dense membrane over the pupillary region. Part of this membrane was removed. This eye was removed elsewhere in 1932. We were unable to obtain the specimen for microscopic examination. The patient remained well and later was married to a man who had normal eyes. She gave birth to a daughter on September 7, 1938,* and another child was born in November, 1940.

Fig. 3 (Benedict and Parkhill). Photomicrograph of tumor of left eye in case 3, showing typical perivascular bands of tumor cells (×40).

Case 4. The patient in this case was a girl, aged two years and four months, who was the first child of the patient in case 3. Early in December, 1940, the patient's grandparents had noticed a change in the appearance of the pupil of her right eye. The child was taken to Dr. Joseph P. Duane, of Peoria, Illinois, who made a diagnosis of bilateral glioma of the retina. In reviewing the literature on this subject, Dr. Duane came across the report of an instance of glioma affecting homologous twins, which had been made previously by one of us[80] (W. L. B.)† As a result of this discovery, he kindly

* This child was the patient in case 4.

† One of these twins (case 3) was the mother of this patient.

referred the patient to the Clinic.

The child was brought to the Clinic by her grandfather on January 16, 1941. The patient appeared to be well nourished. She was very active and had useful vision. Examination of the right eye revealed a white mass which extended forward as far as the lens. In the left eye, the head of the optic nerve was white. On the temporal side of this eye, there was a region of pigmentation which was due to choroidal degeneration. Farther to the temporal side and above the macula, there was a mass which was 4 diopters high and 1½ disc diameters in width.

As the mother of the child had lost both of her eyes because of glioma, she felt that everything should be done to save the life of the child even though it would mean the removal of both of the eyes. Because the position of the tumor in each eye was very close to the optic nerve, it was felt that irradiation therapy would not be helpful. Both eyes were removed on January 24, 1941 (fig. 4).

Microscopic examination of the right eye revealed that, except for a few millimeters anteriorly, the retina had been replaced by a large tumor which extended from the optic disc to the posterior surface of the lens and practically filled the central and nasal portions of the vitreous chamber. The predominant histologic picture was that commonly observed in cases of retinal glioma; that is, perivascular bands or cuffs of living tumor cells surrounded by a mantle of degenerating and necrotic cells which contained occasional regions of calcification. The tumor cells had invaded a small portion of the choroid posteriorly and had penetrated the optic nerve for a distance of about 1 mm. The tumor cells apparently had arisen from the layer of ganglion cells and from the inner nuclear layer and had invaded the other layers. In general, the cells were small and the cytoplasm was scanty. The cells had hyperchromatic nuclei which were spherical, polygonal, or wedge shaped. Toward the periphery of, the tumor, there was a small number of typical rosettes. These consisted of rings of cells arranged about a central lumen which was surrounded by a delicate limiting membrane at the inner borders of the cells. Pseudorosettes were numerous (fig. 5). In these pseudorosettes, the cells were arranged in short arcs or complete circles about clear spaces that were filled with fibrillary material.

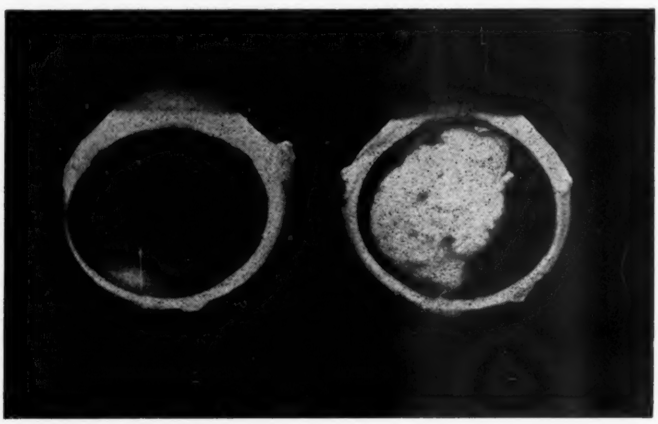


Fig. 4 (Benedict and Parkhill). Gross appearance of bilateral retinal gliomas in case 4. The tumor of the left eye, which was the smaller of the two tumors, was situated close to the optic disc.

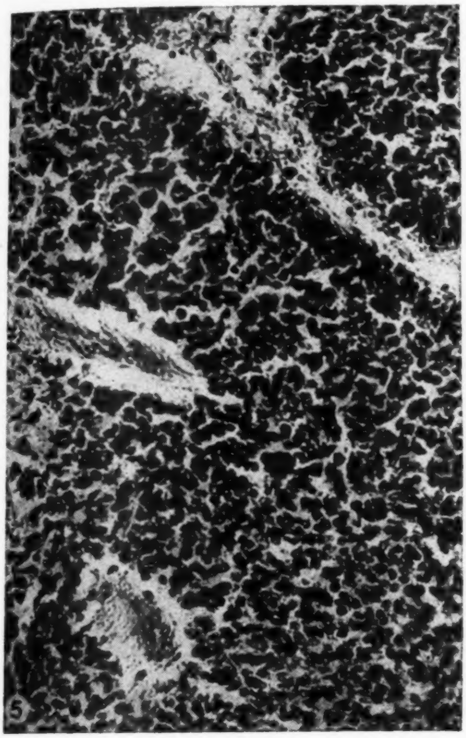
The tumor in the left eye was small (9 mm. in diameter) and it was lentiform in shape. It was situated posteriorly about 1 mm. lateral to the optic disc. Microscopically, it appeared to arise from the region of the layer of ganglion cells and from the inner nuclear layer of the retina. The tumor cells were similar to those seen in the tumor of the right eye but there was little evidence of degeneration and the usual arrangement of the cells into perivascular cuffs or bands was not evident. Rosettes were numerous and pseudorosette formation was extensive (fig. 6). This is the smallest retinal glioma that we have examined and it is the only one in which we have failed to observe the typical perivascular cuffs of tumor cells in at least a part of the tumor. Tumors in both right and left eyes were diagnosed glioma of neuro-epithelioma type.

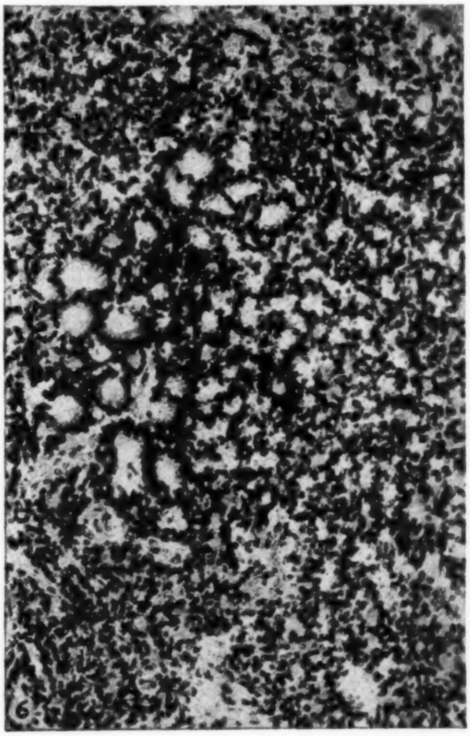
The patient was dismissed from the hospital January 29, 1941, to the care of Dr. Duane. She has not since returned to the Clinic, but was in good condition when last heard from (January, 1943).

COMMENT

The four cases which we have reported in this paper comprise two instances of the occurrence of retinal glioma in successive generations. In case 1, the mother of the patient had had a glioma of the right eye at the age of seven months. In the case of the daughter, the diagnosis was confirmed by microscopic examination. As stated previously, the patients in cases 2 and 3 were twin sisters and the patient in case 3 subsequently gave birth to a daughter (case 4) who had bilateral glioma of the retina at the age of two years and four months. In the four cases the diagnosis was confirmed by microscopic examination. The disease was bilateral in cases 1, 3, and 4. In case 2, there was still no evidence of tumor in the right eye at the time of the patient's death.

In a histologic study of 35 cases of retinal glioma which we reported previously,[61] it was found that the tumors could be divided into two types: (1) a retinoblastoma type, which is highly undifferentiated and resembles the highly malignant medulloblastoma that affects children, and (2) a neuro-epithelioma type, which is partially differentiated and contains rosettes or many pseudorosettes. These rosettes not only are characteristic of the neuro-epithelioma type of retinal glioma but also are present in neuro-epitheliomas of the brain, spinal cord, or peripheral nerves. They represent a partial or slight differentiation of the highly anaplastic tumor cells. Marchesani[62] has suggested that they represent a form of differentiation comparable to that of the primitive neural tube. Pseudorosettes





Figs. 5 and 6 (Benedict and Parkhill). Fig. 5, numerous pseudorosettes in tumor of the right eye in case 4 (×200). Fig. 6, rosettes and pseudorosettes in tumor of the left eye in case 4 (×115).

usually are abundant in gliomas which contain true rosettes. Pseudorosettes apparently indicate partial differentiation of tumor cells which is almost, if not quite, as great as that characterized by the formation of true rosettes.

Good results were obtained in 62.5 percent of cases in which a neuro-epithelioma type of tumor was present, as compared with only 12.5 percent of cases in which a retinoblastoma type of tumor was encountered. It, therefore, appears that the retinoblastoma type of glioma is more malignant than the neuro-epithelioma type. This statement appears to be borne out by cases 2, 3, and 4. In case 2, in which a retinoblastoma type of glioma was encountered, the patient, who was a twin sister of the patient in case 3, died of cerebral extension of the tumor five-and-a-half months after operation. In case 3, in which a neuro-epithelioma type of glioma was present, the patient is alive and apparently is free of recurrence of the tumor 18½ years after operation. In case 4, in which the bilateral tumors were of the neuro-epithelioma type, the patient, who was a daughter of the patient in case 3, was well and apparently had not had a recurrence of the tumor at the time the last follow-up data were obtained.

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ANISOCYCLOPLEGIA*

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In discussions of cycloplegics it seems to be assumed that these agents affect both eyes of the same individual to an identical degree. When mention has been made of persons or races who have ciliary muscles hard to relax, the implication is still that both eyes respond alike. It was with this conception that I and others made studies comparing the effect of one drug on one eye with that of another drug on the fellow eye. Misgivings as to the validity of this procedure were aroused by sometimes finding totally different depths of cycloplegia in the two eyes of the same person under the same circumstances. This would happen to an occasional office patient for whom the same drug was used in each eye. In 1938, I mentioned this phenomenon† and pointed out that homatropine might work well upon one eye of a patient but hardly at all upon the other eye.

Further study indicates that unequal cycloplegia is fairly frequent. The reason for singling out this subject for emphasis is that a review of case reports on refraction submitted as one of the requirements for the certificate of the American Board of Ophthalmology reveals that many of the candidates make a practice of prescribing their cycloplegic acceptance, or even their net retinoscopy. Usually this is done without estimating the residual accommodation. Then an undetected difference of several diopters in the cycloplegia of the two eyes, as *often occurs,* can be misleading. It may account for some bizarre prescriptions.

To illustrate this vagary of cycloplegics a case is here presented in which the difference between the two eyes is not extreme. It is, in fact, rather moderate in degree. The case, however, exemplifies two other aspects of the fickleness of these drugs, directly related to this subject. It is, in the first place, one of those instances in which the noncycloplegic way of estimating the refraction proved much the more reliable. As this somewhat accords with Dr. Lancaster's views, it is particularly appropriate for this publication. It further shows that repeated instillations of cycloplegics in the traditional manner, instead of increasing the effect, may be followed by a paradoxical loss of part of the cycloplegia already induced by the single-drop technique. This patient, furthermore, was by training and intelligence an unusually reliable observer. Finally, her refraction was free from complicating astigmatism. The distance correction was also similar for the two eyes and with it the near points were identical.

Case report

F., aged 24 years, an instructor of nurses in a large hospital, complained of eyestrain at close work.

1. "Dry refraction." Under fogging, her best vision, 6/iv, was obtained with these lenses: R.E. +1.00D. sph.; L.E. +1.25D. sph.

With this correction her eyes saw with equal distinctness. This result was corroborated by retinoscopy with distance fixation, and by a reduction of binocular vision to 6/xii previous to removal of the last +.75D. sph. of overcorrection, according to the cyclodamic technique‡ of Dorland Smith.

* Being portion of a lecture in the Los Angeles Graduate Course, January, 1943.

† Beach. Proc. Postgraduate Course in Ophthalmology, George Washington University, Washington, D.C., 1938, v. 1, p. 90.

‡ Smith, Dorland. Refraction under cyclodamia, Amer. Jour. Ophth., 1926, v. 9, pp. 896-903.

With this distance correction her near point for each eye was six inches, indicating accommodation of 6.50D., somewhat substandard for her age. While this might be due to weak accommodation, it might also result from undercorrection of her hypermetropia.

2. *"Single-drop" cycloplegic refraction.* On the possibility of uncovering more hyperopia, homatropine was instilled. The single-drop method with paredrine was used. At the end of 50 minutes the cycloplegic acceptance was: R.E. +0.37D. sph.; L.E. +0.75D. sph. This was one third of the "dry" correction for the right eye and two thirds for the left.

The near-point test showed: residual accommodation: R.E. 3D.; L.E. 1D. *This discrepancy would indicate that the homatropine had less effect upon the right eye by 2D. than upon the left.*

3. *Conventional cycloplegia.* To avoid suspicion that this inequality of cycloplegia might result from the unorthodox method of administration, the instillation was continued, in the traditional manner, using five repetitions of 2-percent homatropine in each eye. After this the cycloplegic acceptance was: R.E. 0.; L.E. +0.62D. sph. To ascertain whether this inconsistent drop in the acceptance indicated a genuine loss of cycloplegia, the near-point test was repeated. The left eye was unchanged, but the right indicated further reduction of half a diopter of cycloplegia.

Pitfall in prescribing. Now suppose that, after the customary repeated administration of homatropine, the trial-case acceptance had been prescribed, as is often done. Then this patient would have received: O.D. Plano; O.S. +0.62D. sph. instead of the noncycloplegic results: O.D. +0.75D. sph.; O.S. +1.00D. sph.

It will be seen that in the cycloplegic formula the difference between the lenses was two thirds of a diopter, against one fourth of a diopter in the "dry" (and probably more accurate) prescription. Now, while this is not a glaring discrepancy, and might not be disturbing to some patients, it can be characterized only as slovenly refracting. Moreover, much greater aberration is conceivable. Suppose the eyes of this patient had exerted their total reserve, the lenses prescribed would have been out of balance by 2D. Fortunately, as in this case, it is not the rule for eyes under cycloplegia to exercise the major part of their residual accommodation. Just how much, depends on the individual eye. They grade from eyes that struggle vigorously to accommodate to those that seem completely dormant. According to a crude grouping I have attempted,* these eyes fall in the division that try to accommodate, although in this case rather lazily. Yet even this indolent effort was sufficient to unbalance the cycloplegic acceptance.

COMMENT

It is folly to generalize from the small number of case records examined for this study, yet it is probably safe to say that the normal tendency is for the eyes of the same person to be symmetrical in response to cycloplegics. Still, it is risky to trust to this propensity. Out of over 100 cases, there was in 17 a difference in depth of cycloplegia of over one-half diopter. In two cases the eyes differed 7D. and 10D., respectively, in resistance to the drug. As just demonstrated, this discrepancy in near point does not necessarily predicate as extreme a variation in cycloplegic acceptance for distance. Even in the higher cases of latent accommodation a difference of 1.50D. to 2D. in cycloplegic acceptance was large. Yet that is too much to allow to pass unnoticed and much higher variations are conceivable.

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* Beach, S. J. Proc. Postgraduate Course in Ophthalmology, George Washington University, Washington, D.C., 1941, v. 7, p. 26.

As in this case, the method of administration of the drug did not anywhere appear to be a factor. Neither did marked anisometropia have any consistent effect.

Conclusions

The term *anisocycloplegia* is coined to describe a caprice of cycloplegia such that one eye of a person is affected by the drug to a considerably greater degree than the fellow eye. Different methods of administering the drug do not appreciably or consistently alter the situation. The phenomenon appears to be a property of the individual eye, and independent of the method of administration. While it may be deceptive to any examiner taught to rely entirely upon cycloplegics, it is a menace chiefly to those who are accustomed to prescribe either their unverified cycloplegic acceptance, or their retinoscopic findings. It is revealed by the tests for depth of cycloplegia, and by comparison with results of noncycloplegic procedures.

704 Congress Street.

CONCERNING DETACHMENT OF THE RETINA

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In view of the enormous complexity of the entire problem of retinal detachment, it does not seem amiss to record personal observations made during 15 years of experience with the surgical aspects of detachment. The present-day knowledge of this pathologic entity, incomplete as it is, more than suffices to fill a large volume, but still contains large gaps that only individual experiences can fill. Such contributions should be clinical and to the point, and need not be accompanied by reiterated extensive bibliographies.

First, the recitation of a case which, although not entirely unique, is sufficiently rare to justify recording. In July of 1942, a woman, aged 43 years, gave the history of sudden loss of vision in the left eye some two weeks previously. Neither direct nor indirect trauma could be recalled. In the upper quadrant of the lens was a thin, wedge-shaped opacity of the anterior cortex which impeded accurate study of the periphery of the fundus in that quarter. The entire upper half of the retina was detached and billowed forward some 20D. At axis 90° was a fairly large horseshoe-shaped hole in the retina. Operation was advised. She then drove 400 miles by automobile to consult Dr. W. L. Benedict, who confirmed the diagnosis and the advice. The return trip was also made by automobile, after which she entered the hospital. Atropine and binocular bandage were applied, and she was immobilized on her back, with the foot of the bed elevated some eight inches. Within three days the retina had become completely flat and no trace of detachment could be found. The retinal hole, which had been clearly visible, could no longer be seen, partly because of the location, and partly because of the previously mentioned opacity in the lens. Absolute immobilization was continued for three weeks. She was then gradually returned to normal-life activities, and the retina has remained in position. However, the vision in that eye has never been better than 0.3, on account of a cystic condition in the macular area.

In the days before Gonin popularized surgical attack upon detachment of the

retina, it was claimed that as many as 3.5 percent of cases of retinal detachment underwent spontaneous cures (Leber). That statement is open to doubt, and today the surgical results are sufficiently good (average of 50 percent the world over) so that the expectant line of treatment is not justified except in the unusual case. But this does bring up for discussion the question of preoperative immobilization, which has not been given the attention that it deserves. For some years, it has been our habit to immobilize every patient with detachment of the retina in such a position that the most elevated portion of the retina is lowest. As a rule this coincides with the position of the hole, but if it does not, the location of the hole is disregarded. Atropine is used, and both eyes are bandaged continuously except for intermittent examination. This procedure is followed regardless of the length of time that the detachment has existed, and the 5 to 10 days of preoperative immobilization are not considered wasted time that may jeopardize the final result. The resultant decrease in the extent and degree of the detachment appears to have the following significance:

(1) The more rapid and the more extensive the flattening of the retina under immobilization, the better is the prognosis of the proposed surgical attack.

(2) The flatter the retina becomes under immobilization, the more accurate is the localization of the hole in its final position.

(3) The less fluid there is under the retina at the time of operation, the less extensive need be the surgical attack upon the sclera.

(4) The better the approximation of the retina to the choroid, the better are the chances for postoperative firm adhesions and the less the dangers of recurrence of the detachment.

However, a word of caution is necessary. As the retina flattens under the influence of gravity during the course of immobilization, the position of the patient must be maintained until the surgical procedure is completed. This point can best be illustrated by the recital of a case of detachment of the upper half of the retina that became completely flat under four days' immobilization.

Ophthalmoscopic examination revealed a hole well in the upper periphery. The patient was put into a wheel chair to be brought down to the examining room so that the fundus could be sketched and the position of the hole fixed in relation to the retinal vessels. In the 15 minutes that elapsed between being put upright in a chair and being brought to the eye room, the entire upper half of the retina again became detached and billowed forward some 15D. Another two or three days of immoblization brought the retina back into position, after which a successful operation was performed.

Why is it almost impossible to dilate the pupil in some cases of retinal detachment? In a relatively small number of cases, the pupil can never be enlarged to more than about 4 mm. in diameter, regardless of the use of any and all mydriatics. The small pupil, of course, interferes somewhat with the accurate localization of the hole, but does not seem to be of prognostic significance.

In a certain percentage of cases of retinal detachment in which the operation is successful, central visual acuity is not improved to beyond 0.3 or 0.4, due, as Reese has shown, to the formation of a cystic condition in the macular retina. The development of that condition seems to be dependent upon two definite factors: the length of time that the macular retina has been detached from its base, and the

age of the patient. The latter is probably the more important. In peripheral detachment, if the macular area is only slightly or not at all involved, 60 years seems to be the demarcation line between good and only probable chances of success, whereas in detachment of the macular retina, an age between 35 and 40 years forms the deadline. Subsequent to that age, reattachment of the macular area is accompanied in a very high percentage of cases by the cystic formation, even though there is a perfect anatomic reattachment. But under the age of 25 years, detachment of the macular area is not apt to be followed by cystic degeneration, almost regardless of the length of time the retina has been detached. In adults, the cystic formation seems to appear if the macular area has been off for more than about one week. These are not invariable facts, but they do permit of a fairly accurate idea as to the prognosis in any given case.

There are two ways of baring the sclera posterior to the equator in order to expose the area to be attacked. The first is to tenotomize the necessary muscle, thus permitting the eyeball to be rotated as desired, and, upon conclusion of the operation, to suture the cut muscle into the proper position as accurately as possible. The second is to slip a bridle suture under the muscle in question and rotate the eye, more or less forcibly, by traction upon the suture. This second method would appear to be the more logical procedure, for it is obviously impossible to sew a cut muscle back into an anatomically accurate position. But in some mysterious way, a muscle thus cut and sutured becomes finally reattached in its proper place, so that there is no resultant tropia and at most a low degree of phoria. On the other hand, extensive traction upon a bridle suture has in more than one instance resulted in some stretching of the muscle behind the globe (it may be that the bony insertion was displaced) so that there was a resultant permanent tropia. That applies, however, only when excessive rotation of the eyeball is necessary to attack the sclera near the posterior pole.

In using the modified Weve type of operation (baring the sclera over the probable location of the hole—surrounding that area by a ring of partly penetrating scleral punctate cauterizations with the Gradle needle—superficial electrocoagulation of the surrounded scleral area with a Weve flat electrode—final permanent drainage by means of a 0.5-mm. stylet introduced through the sclera with 75 milliamperes of diathermy current), continuous observation of the fundus by indirect ophthalmoscopy must accompany each step. That necessitates a clear cornea throughout the entire course of the procedure. I found that during the latter half of the operation the cornea frequently became so cloudy that a clear view of the fundus was no longer possible. Dr. Snydacker, who was assisting, happened to touch the eye and found that the intraocular pressure was markedly increased, in some instances up to plus 3. Of course that accounted for the corneal edema, which always disappeared within a few minutes after the final perforating puncture had been made. This phenomenon was observed in some 10 or more successive cases. Since then, after the first three or four partly penetrating scleral cauterizations, the subretinal area was drained slowly by three fine needle penetrations of the sclera under 2½ milliamperes of positive catholysis. These punctures are not large enough to evacuate all of the subretinal fluid, but merely allow of seepage, sufficient to prevent the rise in tension. The hypertension is of course due to the shrinkage of the sclera by the diathermy cauterization, with re-

sultant decrease in total volume capacity of the eyeball. The shrinkage is only temporary and the sclera resumes its normal extent within the course of a few days.

One final point which has been stressed repeatedly, but the importance of which has not attained universal recognition, is the necessity of juxtaposition of the retina and choroid while postoperative scars are forming. As Post has shown, it is impossible to obtain reattachment of the retina if there be fluid under the retina that keeps it so far away from the sclerochoroidal areas of cauterization that the retina cannot be included in the formation of the necessary scar tissue. Consequently, it is obvious that thorough drainage of the subretinal space is an essential, and this is possible only with an adequate-sized hole in the sclera that will remain open for at least three days. One operator prefers to make such a hole with a trephine, while another prefers a penetrating stylet puncture made with diathermy current. But certain it is that a simple scleral puncture, made with a Graefe knife, will close in 24 hours so that no further drainage is possible.

And finally come the questions as to how long after operation the patient should be kept quietly in bed and how long should hole spectacles be worn? Neither of these has as yet been answered authoritatively. Acting under the influence of the Gonin teachings, we say absolute rest in bed for 14 days, up in wheel chair with limited activity for the subsequent 10 to 14 days, moderate amount of freedom at home for another four weeks, and finally return to a sedentary occupation some three months after operation. Whether or not such procedure is unnecessarily harsh we are unable to say, for none of us dare experiment with a successful outcome after operation. Hole spectacles with a 3- or 4-mm. aperture are ordered to be worn for at least two months, after which the size of the aperture is increased every three weeks until the diameter is 1½ cm. This latter size is worn up to five or six months after operation. This also may be unnecessarily long, but it seems to be the safest procedure.

58 East Washington Street.

RESECTION OF THE LEFT INFERIOR OBLIQUE MUSCLE AT ITS SCLERAL ATTACHMENT FOR POSTOPERATIVE LEFT HYPOTROPIA AND LEFT PSEUDOPTOSIS*†

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Underaction of a muscle is usually accompanied by spasm of the synergist of the other eye, and in many cases it is desirable to attempt to weaken the action of the overacting muscle. The most common example of this procedure is myotomy or retroplacement of the spastic inferior oblique of one eye for parèsis or paralysis of the superior rectus of the other eye. However, when underaction of a vertical muscle is associated with ptosis, caused by the depressed position of the eyeball, as may be the case in postoperative paresis of the inferior oblique, weakening the action of the spastic muscle will not correct the ptosis. Therefore, an operation which will elevate the lower eye and secondarily the eyelid should be selected.

In 1935, Wheeler[1] proposed shortening the inferior oblique by tucking the muscle just behind its origin or by anchoring the tuck to the periosteum of the orbital margin. He found this procedure especially useful in the treatment of hypophoria associated with ptosis and suggested that this operation may be combined with an advancement of the superior rectus of the other eye.

In this paper resection with advancement of the inferior oblique at the scleral insertion is described for the correction of paresis of the inferior oblique with marked hypotropia and secondary pseudoptosis.

TECHNIQUE

Under local or general anesthesia, tincture of metaphen and alcohol are applied to the eyelids. The eye speculum is inserted and the cornea is covered with a fine film of sterile petrolatum. A traction suture of black silk is introduced in the episcleral tissue near the limbus at approximately the 4-o'clock position. This suture is pulled toward the upper and medial angle of the orbit.

With a Stevens scissors, a curved incision is made in the conjunctiva, extending from a little above the outer canthus to a point deep within the cul-de-sac over the inferior rectus (fig. 1 *a*). Tenon's capsule is grasped with a mousetooth forceps just below the lateral rectus. The capsule is opened with scissors, and the wound is enlarged slightly downward. The traction on the limbal suture is replaced by direct traction by means of a double scleral hook.[2] The tendon of the inferior oblique is seized by means of a fixation forceps with a catch. To permit this, the normal adhesions between the lateral rectus and the inferior oblique may have to be freed.

The scleral insertion of the inferior oblique is exposed, and the muscle is grasped with a special muscle forceps at a distance from the scleral insertion equal to the amount of resection desired (fig. 1 *b*).

A double-armed nylon 6-0 suture is introduced from the scleral surface of the proximal part of the muscle through the depressions of the special forceps. The suture is also passed through the episclera

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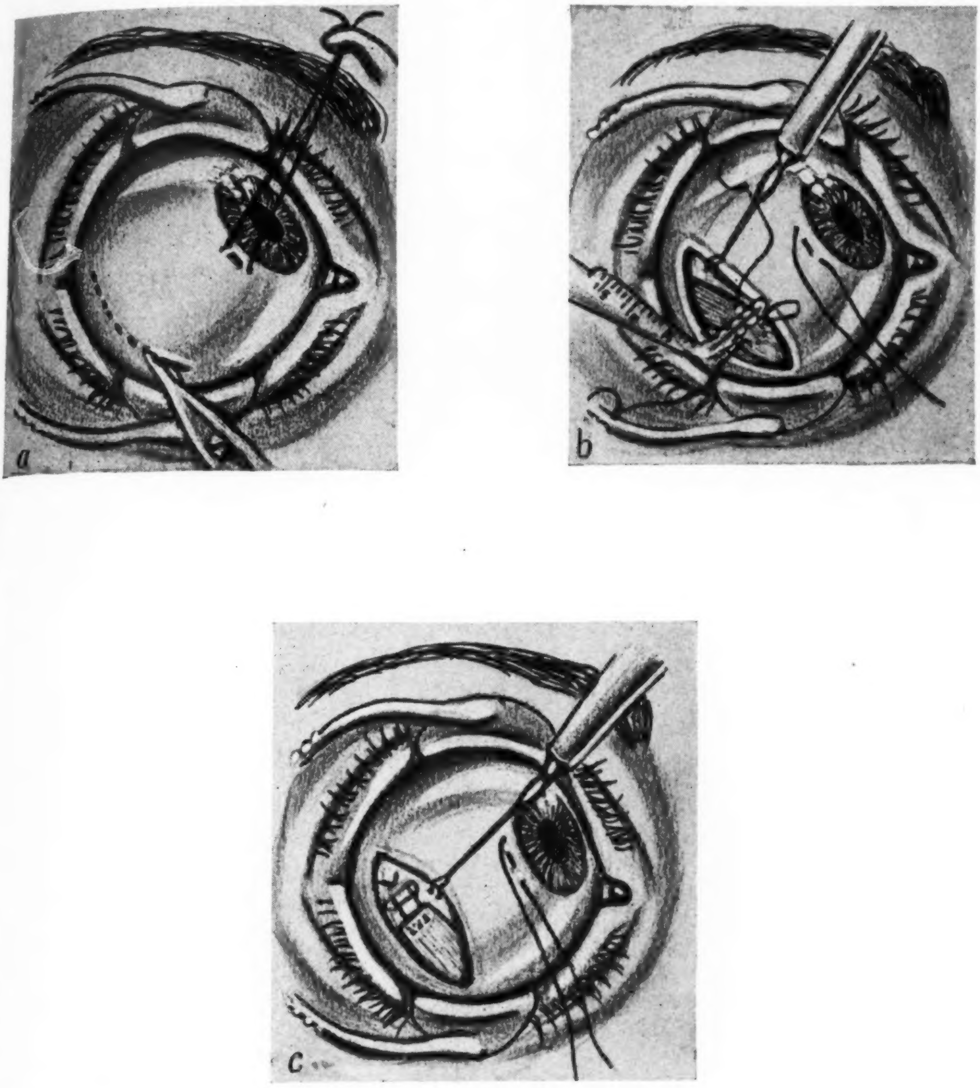

Fig. 1 (Berens and Loutfallah). Steps in resection of the left inferior oblique muscle. *a,* a curved incision is made in the conjunctiva with a Stevens scissors, starting a little above the outer canthus and ending in the cul-de-sac. *b,* the muscle is grasped with a special muscle forceps at a distance from the scleral insertion equal to the amount of resection desired. A double-armed suture is introduced. *c,* the suture is also passed through the episclera above and close to the anatomic insertion of the muscle. Traction by means of the scleral hook is relaxed while the suture is tied.

above and close to the anatomic insertion of the muscle.

The part of the inferior oblique between the clamp and the sclera is resected cautiously with careful avoidance of the nylon suture just placed. The special forceps is removed (fig. 1 *c*).

The traction by means of the scleral hook is relaxed while the suture is tied securely with a surgeon's knot followed by a single loop. The free ends are cut close to the knot.

The conjunctival incision is closed, using a single 5-0 plain catgut suture on an

atraumatic needle. Metaphen ointment (1:3,000) is inserted between the eyelids and a dressing is applied.

When the resection involves a large piece of the muscle, the incision of Tenon's capsule may be advantageously performed above the lateral rectus in the superolateral quadrant. Although we have not used this procedure for the correction of anomalies of the inferior oblique, this modification of technique seems advisable in some instances. In such a case the conjunctival incision is extended above the lateral rectus.

Case report

Miss G. W., aged 14 years, was first seen on December 6, 1941. She gave a history of reading difficulties, which had been present for some time, associated with a convergent squint and ptosis of the left upper eyelid. There was a marked tilting of the head toward the right shoulder.

There was no history of squint in the family. The patient had frequent colds, and for years had had recurrent abscesses of the ear. At the age of four years an operation was performed on the cervical glands. She had worn correcting lenses for four years in an attempt to correct an esotropia of 55^{Δ} for distance and 45^{Δ} for near; apparently there was an upshoot of the eyes in adduction, which was more marked in the left eye.

On November 13, 1934, a resection of the left lateral rectus, a 4-mm. recession of the left medial rectus, and a tenotomy of the left inferior oblique had been performed by another surgeon. Postoperatively the findings* with the right eye fixating were: without correction S & P

E' 35^{Δ}, RH' 8^{Δ} for distance
E'' 40^{Δ}, RH'' 8^{Δ} for near

with correction S & P

E' 13^{Δ}, RH' 8^{Δ} for distance
E'' 20^{Δ}, RH'' 8^{Δ} for near.

With correction visual acuity of the right eye was 20/20 and the left eye 20/40 plus. The synoptophore objective readings were esotropia of 15^{Δ}, with right hypertropia of 10^{Δ}. Approximately a year after the operation the cosmetic result was apparently satisfactory, but when the left eye was adducted, elevation was limited. There was no marked change in the deviation.

In November, 1937, three years after the operation, the muscle balance was recorded as follows: without correction

E' approximately 30^{Δ} RH' 20^{Δ} for distance
E'' approximately 30^{Δ} RH'' 20^{Δ} for near

with correction

E' 2^{Δ} RH' approximately 20^{Δ} for distance
E'' 6^{Δ} RH'' approximately 20^{Δ} for near

Our first examination was performed on December 6, 1941. The following findings were noted: Vision without correction, right eye 20/15, left eye 20/30. Muscle balance without correction

E' 40^{Δ} RH' 18^{Δ} for distance
E' 43^{Δ} RH'' 30^{Δ} for near

with correction

E' 15^{Δ} RH' 12^{Δ} for distance
E'' 25^{Δ} RH'' 28^{Δ} for near

There was a pseudoptosis of the left upper eyelid, and scars were present in the conjunctiva over the medial and lateral recti of the left eye. Although the patient could appreciate diplopia, suppression of the left eye was possible. The patient was unable to fuse the Worth four-dot test and could alternate fixation at will.

On December 19, 1941, the screen test with prisms in the cardinal positions of gaze gave the following measurements at 75 cm. without correction:

* The abbreviations used signify: S & P—screen and parallax; E'—esotropia; RH'—right hypertropia; '—at 25 centimeters.

	Right		*Left*	
(up)	E^t 18$^{\Delta}$		E^t 14$^{\Delta}$	(up)
	RH^t 25$^{\Delta}$	Primary Position (75 cm.)	RH^t 30$^{\Delta}$	
	E^t 15$^{\Delta}$	E^t ....27$^{\Delta}$	E^t 25$^{\Delta}$	
	RH^t 25$^{\Delta}$	RH^t ....20$^{\Delta}$	RH^t 30$^{\Delta}$	
(down)	E^t 10$^{\Delta}$		E^t 23$^{\Delta}$	(down)
	RH^t 16$^{\Delta}$		RH^t 24$^{\Delta}$	

In addition to these findings nystagmoid movements were present when looking to the right, but were absent when looking to the left. When looking up and to the left there was weakness of the left superior rectus and a spasm of the right inferior oblique. When looking down and to the left, there was a slight overaction of the right superior rectus. To correct this marked spasm of the right inferior oblique and the residual esotropia, a 10-mm. myectomy of the right inferior oblique and a 3.5-mm. retroplacement of the right medial rectus were performed on December 31, 1941. Postoperatively the condition was slightly improved.

On January 9, 1942, the muscle balance at 6 M. was: with correction E^t 20$^{\Delta}$, RH^t 10$^{\Delta}$. The ptosis of the left upper eyelid was practically unchanged.

On February 23, 1942, the muscle balance in the cardinal positions of gaze at 75 cm. was as follows:

	Right		*Left*	
(up)	E^t 18$^{\Delta}$		E^t 24$^{\Delta}$	(up)
	RH^t 27$^{\Delta}$	Primary Position (75 cm.)	RH^t 7$^{\Delta}$	
	E^t 20$^{\Delta}$		E^t 23$^{\Delta}$	
	RH^t 22$^{\Delta}$	E^t 28$^{\Delta}$	RH^t 13$^{\Delta}$	
		RH^t 12$^{\Delta}$		
(down)	E^t 16$^{\Delta}$		E^t 25$^{\Delta}$	
	RH^t 16$^{\Delta}$		RH^t 15$^{\Delta}$	(down)

At this time the patient complained of tilting of the image of the left eye and incyclotropia ($-8°$) was diagnosed.

On July 14, 1942, a 6-mm. resection of the right lateral rectus and a 7-mm. resection at the scleral attachment of the left inferior oblique were performed. Ten days after the operation the findings at 6 M. were: with correction S & P—X^t 6$^{\Delta}$, RH^t 6$^{\Delta}$. The patient was able to fuse for distance, and there was second-grade fusion with true correspondence. The head tilt was no longer present, and the pseudoptosis was corrected.

On August 27, 1942, the screen test with prisms in the diagnostic positions of gaze at 75 cm. revealed the following measurements:

	Right		*Left*	
(up)	RH^t 35^{Δ}	Primary Position (75 cm.)	0	(up)
	X^t 8^{Δ}	E^t 18^{Δ}	0	
	RH^t 30^{Δ}	RH^t 8^{Δ}		
(down)	RH^t 16^{Δ}		E^t 20^{Δ}	(down)

On September 3, 1942, the muscle balance at 6 M. was: without correction E^t 35^{Δ}, RH^t 7^{Δ}; with correction $E^{t'}$ 4^{Δ}, RH^t 6^{Δ}. On the orthoptoscope there was second-grade fusion without amplitude at the following angle: E^t 22^{Δ}, RH^t 4^{Δ}, incyclotropia 4°.

Prisms were added to her correction:

R.E. + 2.00D. sph. $\subset\supset$ + .50D. cyl. ax. 90° $\subset\supset$ ½$^{\Delta}$ base down
L.E. + 2.50D. sph. $\subset\supset$ + .75D. cyl. ax. 50° $\subset\supset$ 8^{Δ} base up

With this correction binocular vision was maintained, but occasionally a blinking movement was necessary to overcome diplopia when convergence and accommodation were modified. However, tilting of the retinal images was more apparent to the patient.

Discussion

The full correction of hypertropia and hypotropia is an important factor in the successful surgical treatment of the combined heterotropias. According to Fuchs, the scleral insertion of the inferior oblique muscle is the most variable of all the extraocular muscles. This fact suggests that the inferior oblique may often require surgical correction.

To oppose the lengthening operations, which are indicated in cases of spasm or overaction, a shortening operation should be considered when paresis or underaction is present, especially if pseudoptosis caused by hypotropia is a factor. Hypotropia when dominant in certain fields and especially when associated with a homolateral pseudoptosis should be corrected by resection of the underacting muscle and not by weakening the contralateral synergist. It is obvious in this case that resection of the left inferior oblique was necessary in order to correct the hypotropia. Retroplacement of the right superior rectus, which may correct the residual hypertropia, is now contemplated.

As to resection of the inferior oblique, there is no specific rule for the evaluation of the amount of resection. In this case a 7-mm. resection corrected 10^{Δ} of hypotropia in the field of maximum action of the muscle and only 4^{Δ} in the primary position. The residual cyclotropia of 4 degrees might have been corrected by a larger resection, and Jackson's suggestion of displacing the rectus insertion[3] may have to be considered if the cyclotropia continues to be troublesome. At present the cyclotropia, although less in degree, is more troublesome, especially when the images are approximated with vertically placed prisms.

It would seem advisable to attempt to correct postoperative cyclotropia by shortening the weakened muscle if the weakness seems purely the result of pre-

vious surgery. In the case reported here, tenotomy at the orbital margin had been performed and, therefore, resection at the scleral insertion was considered advisable. Study of the literature has revealed no previous report of resection of the inferior oblique at the scleral insertion.

The greatest difficulty lies in selecting proper cases for resection of the inferior oblique. This should be determined after thorough and repeated examinations, including: careful refraction, fields of monocular rotation or fixation, diplopia fields when possible, determination of the muscle balance in the cardinal positions of gaze, the screen-comitance test, and study of fusion, fusion amplitude, and cyclotropia.

Summary and Conclusions

A simple method of resection with advancement of the inferior oblique muscle at the scleral insertion is described. It affords a means of correcting hypotropia, overcoming pseudoptosis, and improving cyclotropia, resulting from underaction of the inferior oblique.

Two approaches to the scleral insertion of the inferior oblique are suggested for this operation—either below or above the tendon of the lateral rectus. The resection is greatly facilitated by the use of a scleral hook, which assists in the exposure of the insertion, and a calibrated muscle forceps, which permits rapid and accurate estimation of the amount of resection. A case of paresis of the inferior oblique with left hypotropia of 20^{Δ} in the primary position with an associated esotropia, pseudoptosis of 2 mm. and 8° of incyclotropia is reported. The postoperative diagnosis was a right hypertropia of 8^{Δ}, with a residual esotropia of 22^{Δ} and incyclotropia of 4°, while the pseudoptosis was completely corrected. As the right hypertropia seems to be increasing, retroplacement of the right superior rectus is being considered.

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VENESECTION IN A CASE OF CHOROIDAL HEMORRHAGE FOLLOWING CATARACT EXTRACTION

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The only purpose in presenting this case report is to inform the profession of a simple procedure that on one occasion seemed effective in a case, which, based upon past history, observation for six months prior to operation, and brisk bleeding from a point below the iris level following a combined extracapsular extraction, must be classified as a choroidal hemorrhage.

Many average and some excellent ophthalmic surgeons must have met with this most serious complication more often than would appear from reports in the literature; for this reason, it seems that a short reference to the literature, as reviewed so well by Ziegler, would be an appropriate preface.

Dr. S. Lewis Ziegler reviewed the subject for the Jackson Birthday Volume in 1926—officially entitled "Contributions to Ophthalmic Science." He cited 131 references covering the period from 1786 to 1924. He made four classifications, or forms, of the manner in which "the pathologic lesion" had been observed, as follows:

1. As a slight ecchymosis which von Graefe first observed.
2. As broad, flat effusions with or without chorioretinal detachment.
3. As punctate or papillary subchoroidal hemorrhages.
4. As a true expulsive hemorrhage of the choroid in which the choroid and retina are either ruptured or torn loose from the sclera, the venous blood flowing freely and forcing out all the contents of the globe.

Thirteen possible causes of choroidal hemorrhage are listed and evaluated, based upon clinical observation and pathologic studies of laboratory specimens.

Etiologic factors considered were five in number:

1. Arteriosclerotic changes, either local or general.
2. Sudden lowering of the intraocular tension through decompression of the globe.
3. Traumatic detachment of the choroid through surgery, contusion, or strain.
4. Disturbance of the sympathetic nervous system through emotional shock.
5. A hemophilic tendency.

Ziegler reported that Fage,[1] Knapp,[2] Spalding,[3] and others have called attention to the fact that the hemorrhage in their cases "came from the anterior choroidal vessels, just back of the ciliary body." Because the case here reported was of this type, it is presented with the hope that the simple postoperative procedure, which proved so effective, may be of value to the profession.

R. G. S., a white man, aged 56 years, occupation, chef, came for examination on January 16, 1930.

Diagnosis. Immature cataract O.D.; anophthalmos O.S. (surgical). The patient knows that he has had diabetes for the past five years, and arterial hypertension for the past three years. For the past four years his vision had been failing, more markedly in the left eye. This eye was operated on for cataract three years ago, and later enucleated "because of hemorrhage."

External examination. The right eye

was white and quiet; the cornea and aqueous were clear; the pupil reacted promptly to light; the tension was normal. Vision sufficed only to detect fingers at 6 feet, although a good visual field was present. Ophthalmoscopic examination revealed a good reflex, and the vitreous apparently clear.

The patient was referred to an internist, who kept him under observation until July 28, 1930, when he made the following report:

January 20, 1930. Blood pressure 180/100; moderate arteriosclerosis; diabetes; blood sugar, fasting 190 mg. per 100 c.c. of blood. Diet: High carbohydrates and low fat.

On *July 30, 1930,* the patient was admitted to the hospital upon advice of the internist, who reported: Urine 0.1 percent sugar; blood sugar 160 mg. Patient considered safe for surgery. Hospital laboratory reported: Urine sugar ++, sediment negative; blood sugar 180 mg.; lens protein 1 c.c. intradermally showed no wheal in 30 minutes (slight erythema). Operation postponed. Patient discharged.

On *October 30, 1930,* the patient was again admitted to the hospital. The diagnosis was: Mature cataract, O.D. Laboratory report: Urine straw colored, acid; specific gravity, 1,028; albumin 0, sugar 0, blood sugar 100 mg.; blood pressure 160/80.

On the following day the right eye was operated on under local anesthesia. A scleroconjunctival suture was placed, and corneal section, iridectomy, capsulotomy, and lens delivery were effected without undue manipulation; the suture was tied, and the iris reposited.

At this point, just as the anterior chamber was about to be irrigated, a brisk flow of yellowish blood was observed coming from below the iris near the surgical coloboma. The operating room nurse was sent to the telephone (outside the operating room) to call the internist, who was on duty at the time in the medical clinic of the Massachusetts General Hospital (same building), and advise him of the emergency. He responded promptly—during which time atropine and White's ointment were instilled into the conjunctival sac and a double dressing was applied.

The internist (R. P. D.) withdrew 22 ounces (660 c.c.) of blood from a vein in the arm (the amount being left to his discretion), and the patient experienced no discomfort in the eye.

Upon first postoperative examination, approximately 26 hours later, the aqueous was clear and the eye white and quiet. There was no evidence of intraocular hemorrhage.

On *December 3, 1930,* a discission was performed upon the eye. Six days later, with correcting lens, the patient had vision of 20/30 and later vision of 20/24 (with finished prescription).

On *May 6, 1932,* vision O.D. was 20/15; the fundus was essentially negative. On *October 14, 1932,* vision, O.D., was 20/15, with slight change of correction. On *April 14, 1936,* vision, O.D., was 20/20+, with slight change of correction. A report from the patient's daughter on *February 1, 1943,* stated: "Vision as good as ever."

It would seem that Ziegler's fourth classification of choroidal hemorrhage should be further subdivided into (a) the less violent, even trickle of blood from a level below the iris following iridectomy or lens extraction, and (b) the expulsive type, which offers little or no opportunity for therapy of any kind.

Venesection, however, appears to be a safe and simple procedure for checking the former when carried out immediately.

The favorable result obtained in this case suggests that other surgical problems,

such as separation of the choroid following cataract extraction and hyphema requiring irrigation to prevent glaucoma and for blood staining of the cornea, might be treated similarly.

520 Commonwealth Avenue.

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[2] Knapp, H. Arch. of Ophth., 1891, p. 78.
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SOME CLINICAL APPLICATIONS OF FLUORESCENCE IN RELATION TO MELANOTIC PIGMENT

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The striking demonstration of ordinarily invisible freckles on a person's face accidentally illuminated by ultraviolet and very short violet rays led to a limited survey of the possible value of the so-called Black-light in clinical ophthalmology* (figs. 1 and 2). Although in the literature mention is made of this observation, it does not appear to be well known. Goodman[1] however, pointed out that freckles, even in persons who show few or none under ordinary light, stand out markedly in their full brown color under conditions proper for producing fluorescence. He observed that old faded spots of "sunburn" could be seen as dark areas contrasting with adjacent normally fluorescing areas of "unsunburned" skin. He also reported having found that skin melanomata, including a few unpigmented lesions, appeared strikingly dark. It was his observation that the Negro skin normally did not fluoresce but could be made to fluoresce after exposure to actinic light for a time sufficient to produce an erythema in a Caucasian. However, the negroid skin normally does appear different from freckled areas in a Caucasian when viewed under filtered ultraviolet light.†

Interpretation of these findings by terse statements is not easy because certain necessary details apparently are not known at this time. One must know if the normal fluorescence of Caucasian skin for the most part normally arises at the depth or deeper than that of the position of freckles in which case the freckles could simply hide the usual luminescence. However, the freckles have a definite brown color and are not simply areas of the skin that do not fluoresce. One must also know whether freckles and unpigmented melanomata appear brown because their presence alters one or more of various characteristics, such as color, translucence, and character of fluorescence of the superimposed layers, so that

* As the greatest value at this time appears to be related to demonstration of obscure melanin, only mere reference is made here to the graphic demonstration of preclinical pinguecula. The possible use on differentiation of hypopyon in the anterior chamber from free necrotic tumor cells was attempted without satisfactory result.

† Unless otherwise specified below, all mention of *filtered ultraviolet light* refers to light from a G.E. Mazda 100-W. Mercury Projector Spot bulb passed through a Corning Glass filter, 5D. CVX. RDL. 6 mm. thick, in an otherwise darkened room.

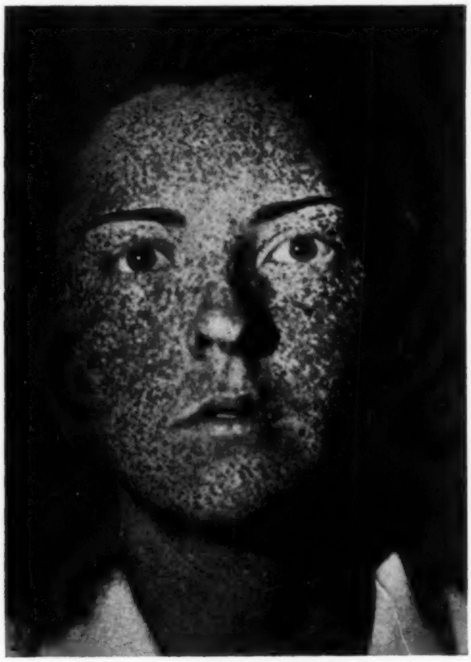

Fig. 1 (Terry). An unretouched photograph of a subject, without cosmetics, taken under photo flood diffused light. Because of the ultraviolet rays from the light, freckles show up slightly more than under ordinary artificial light or daylight. The freckle pattern, including three on the mucous membrane of the upper lip, can barely be seen. The subject during childhood had many very obvious freckles which gradually grew less evident as she matured.

Fig. 2. The same subject as in figure 1, photographed under the rays of a mercury-vapor lamp passed through an ultraviolet Corning Glass filter, 5D. CVX. RDL. 6 mm. thick. The color of the freckles is a rich, dark brown but it is not so deep a brown as is seen in even unpigmented skin melanomata viewed under the same conditions.

the freckles are seen in their full color in contrast to areas of brightly fluorescing pigment-free skin. If melanin and premelanin do fluoresce under ultraviolet light to produce the effect observed, intensity of the fluorescence must then be sufficient to overshadow that of the superimposed keratin and epithelium. Again, fluorescence, in general, is not limited to exposure to ultraviolet light alone because fluorescein in dilution fluoresces brightly under almost any form of artificial illumination, a property that makes it so valuable in clinical ophthalmology. Thus freckles may owe their color under ordinary illumination to very bright fluorescence. The fading of freckles as one grows older may represent truly a partial bleaching or reduction in melanin; however, development and maturity of the superficial layers of the skin may reduce transparency so that freckles may only appear to fade. In whichever case, filtered ultraviolet light can demonstrate them vividly and also reëxposure to a large amount of actinic light can cause a redevelopment of pigment or a return to more youthful transparency of the overlying skin layers. The freckle pattern appears to be constant through life, according to the limited data at hand. If freckles do bleach or fade and redevelop, then there is some basis for the belief that actinic light may have a stimulating effect on the growth of malignant melanotic neoplasms. No attempt is made to explain

these contradictory viewpoints except to dwell on the finding and their importance in clinical examination.

The study of tissues having a potentiality of pigmentation is startling but not invariably consistent. In one patient widespread metastases of a choroidal melanoma proved especially interesting. Of four unpigmented metastases to the skin of the scalp, three appeared heavily pigmented under filtered ultraviolet illumination and could not be distinguished from a fifth metastasis that appeared heavily pigmented under ordinary lighting. The darker, richer brown color of the unpigmented and pigmented skin metastasis was more striking than the color of freckles demonstrated in a person apparently freckle free. For cosmetic purposes these metastases were removed. The cut surfaces of these specimens appeared heavily melanotic only under the filtered ultraviolet light. Microscopic study confirmed the clinical diagnosis. This "fluorescence" of an unpigmented melaninlike substance in a true melanotic color was evident even in mounted, stained sections of the tissues, this brown color overshadowing the fluorescence of the cover slip, of the mounting fluid, of the embedding material, and of the stains used. The patient's urine was heavily loaded with melanin, as shown best by the ferric chloride test.* However, the untreated urine fluoresced in the normal color, thus no evidence of a melaninlike substance was obvious from the test for fluorescence alone.

These observations were repeated while using the Hildreth carbon-arc lamp.[2] The inferiority of the arc lamp to the mercury-vapor lamp as a source of the filtered ultraviolet light was striking. In the first place, when the arc-lamp was the source, freckles, obvious or obscure, unpigmented nevi and melanomata, took on a purple-red color indistinguishable from hyperemia, and hemorrhages near the surface; whereas, although the purplish color of varicose veins, hemangioma, and blood in any manner was unchanged under the mercury-vapor lamp as the source of filtered ultraviolet light, the parts containing a melaninlike substance took on the characteristic deep, rich brown color. A study of the appearance of skin containing hidden freckles was made under each light source and viewed through a yellow filter which would absorb visible violet rays reflected from the surface examined. Some visible violet rays passed through all ultraviolet filters placed before the light source. No effect on the color of the freckles was observable with the mercury-vapor lamp, whereas the reddish freckles seen with the carbon-arc lamp disappeared completely. This difference between the fluorescences from the two light sources was evident when any one of three different kinds of ultraviolet filters was placed between the light source and the skin. It appears safe to conclude that the purplish color of the freckles seen under the arc lamp is a reflection of the visible violet rays that did pass through the lamp filter, representing perhaps a demonstration of the freckle pattern through lack of skin fluorescence in this region, according to the theory presented in the second paragraph. The mercury-vapor lamp gives off 1,800 A.U. to 14,000 A.U.,[3] and, although the iron-cored carbon arc gives off wave lengths as short as 1,800 A.U.[3] also, the common iron-free carbons, such as those available for this study, did not give short-wave lengths of sufficient quantity to produce the same effect as the mercury-vapor lamp.

Use of the ultraviolet light is of definite value in diagnosing unpigmented nevi and

* The melanuria was more evident by this test when freshly prepared 5-percent ferric chloride solution was used on urine 12 to 24 hours old.

in determining their size. They almost invariably show up under filtered ultraviolet light in a true melanin color, and both unpigmented and pigmented nevi under conditions proper for producing fluorescence appeared to be considerably larger than when viewed in ordinary illumination because of the fluorescence. In fact it gave strong suggestion that few if any nevi of the conjunctiva are completely removed unless the ultraviolet light is used as a guide. That, in turn, discourages surgical removal since one is not likely to remove such large areas of conjunctiva as are commonly involved.

Summary

1. Why hidden or obscured freckles in the skin or conjunctiva are so dramatically demonstrated under filtrated ultraviolet light in the true, rich brown color of melanin is not obvious.

2. Relatively unpigmented tissues containing potentiality of pigmentation, such as unpigmented melanomata and unpigmented nevi, almost invariably appear to be heavily loaded with rich brown pigment under filtered ultraviolet illumination from the mercury-vapor lamp, a finding of perhaps real clinical value.

3. Under conditions proper for fluorescence, unpigmented nevi usually appear too large to be removed completely, in many instances discouraging attempts at excision.

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LANCASTER'S TECHNIQUE OF CATARACT EXTRACTION*

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Modifications in technique of cataract extraction continue to be advocated and to appear in the literature. It would seem that the major variations would have been long since exhausted. Nevertheless, ophthalmologists continue to spend considerable time in working out new techniques, and fortunately so, as is proved by Dr. Walter B. Lancaster's technique, which I have found such a safe procedure that I particularly recommend it to young ophthalmologists who need to utilize every precaution to insure success in their first operative cases.

I was fortunate in having been associated with Dr. Lancaster while he was Chief of Staff of the Dartmouth Eye Institute. It was his desire to forego an active part in the surgery in his post, but not before he had taught his associates what he, after 50 years of experience, considered the safest technique of cataract extraction—safe enough so that one could reasonably assure (though not guarantee) the patient a good result.

It was quite clear from the beginning that Dr. Lancaster's was a technique of hands as well as of brains, and that for good work in cataract extraction, capable hands and flair constitute great assets. No two cases can be standardized, and knowledge and judgment should come to the aid of the eye and hand. Each case is unique, and one should be quick to appreciate how to vary the procedure according to the nature of the case and to anticipate any probable trouble during convalescence.

I shall never forget my talk with Dr. Lancaster following my first cataract extraction at Dartmouth. Surgery there is done in a general hospital, and, naturally, I found the experience a departure from that gained in my recent residency at the Wills Hospital, where only ophthalmic surgery is performed. Dr. Lancaster asked me whom I considered to be the finest surgeon, other than an ophthalmic surgeon, I had ever seen operate. I cited two who came quickly to mind. Both, he pointed out, were never known to operate except with everything in perfect readiness for the operation; and one of them, he said, was never known to operate outside of his own clinic. His point was inescapable; namely, that the ophthalmic surgeon should always have everything in as perfect order as possible before operating.

Young ophthalmologists should seize every opportunity to watch skilled surgeons operate. In time one cannot help but acquire something from the skilled surgeon that becomes a part of one's own technique.

As a teacher, Dr. Lancaster did not expect his procedure to be adopted by me in every detail. The most important thing was that I understand the mechanics of each procedure. Naturally, a good operator does his best work with instruments with which he is fully familiar, although he should be able to do a good extraction even with instruments which were not intended for the purpose. Minor differences in technique have always been of interest to Dr. Lancaster as a surgeon, even though he may have marked prejudices on major or minor points. The true value of Dr. Lancaster's technique, as he has taught it to me, lies in its usefulness to the average ophthalmologist and in its benefit to his patients.

* From the Dartmouth Eye Institute.

Anesthesia

PRELIMINARY OR "BASAL" ANESTHESIA

Poor results in cataract surgery are frequently attributed to poor coöperation on the part of the patient. Dr. Lancaster has for years held the view that poor coöperation is preventable, and the fault of the surgeon. His procedure, I believe, completely controls the patient's reactions.

To allay the nervousness felt upon entering the hospital, the patient is given 1½ grains of nembutal, seconal, or phenobarbital, or 3 grains of sodium amytal. A dose is given at 8 p.m. each night before operation, to insure a good night's rest.

A cleansing enema may be given the night before if the operation is to be performed in the morning. If the operation is scheduled for the afternoon the enema should be given about 7 a.m. A light breakfast and liquids are given at noon.

One-and-one-half hours before the operation, grains 20 to 30 of chloral hydrate are given by rectum.

One hour before the operation, 6 grains of sodium amytal are given by mouth, followed by a copious hot drink.

One-half hour before operation, 2 drops of 4-percent euphthalmine or 1-percent paredrine are instilled into the eye to be operated on. Dr. Lancaster objects to the use of atropine before the operation. In fact, should a too zealous resident or nurse instil atropine before the patient is taken to the operating room, he would postpone the operation for a week or ten days. He considers that the use of atropine favors prolapse and that even though eserine is used it only temporarily overcomes the action of the atropine.

The size of individual doses is based upon several things. One should inquire from both patient and nurses concerning the effect obtained from the sedatives previously administered. The size and age of the patient are important factors; but most of all one should consider the nervous system of the patient, and need not hesitate to give a larger dose to the excited or apprehensive patient.

To help produce the sedative effect desired, the patient should be kept quiet, with no visitors or activity in the room.

The patients upon whom I have operated following this routine have been quiet, relaxed, and placid. In instances in which the desired effect is not produced, Dr. Lancaster recommends a hypodermic of 1/150 to 1/200 hyoscine, combined, if necessary, with one-eighth grain of morphine. This should produce the desired effect and will not cause nausea.

Dr. Lancaster's extensive experience, to which I can add my own, stresses the wisdom of this careful preliminary treatment and the use of several drugs in moderately large doses instead of one drug in a very large dose. The activities of the reflex centers are reduced and the patient does not want to move even if he could.

LOCAL OR TOPICAL ANESTHESIA

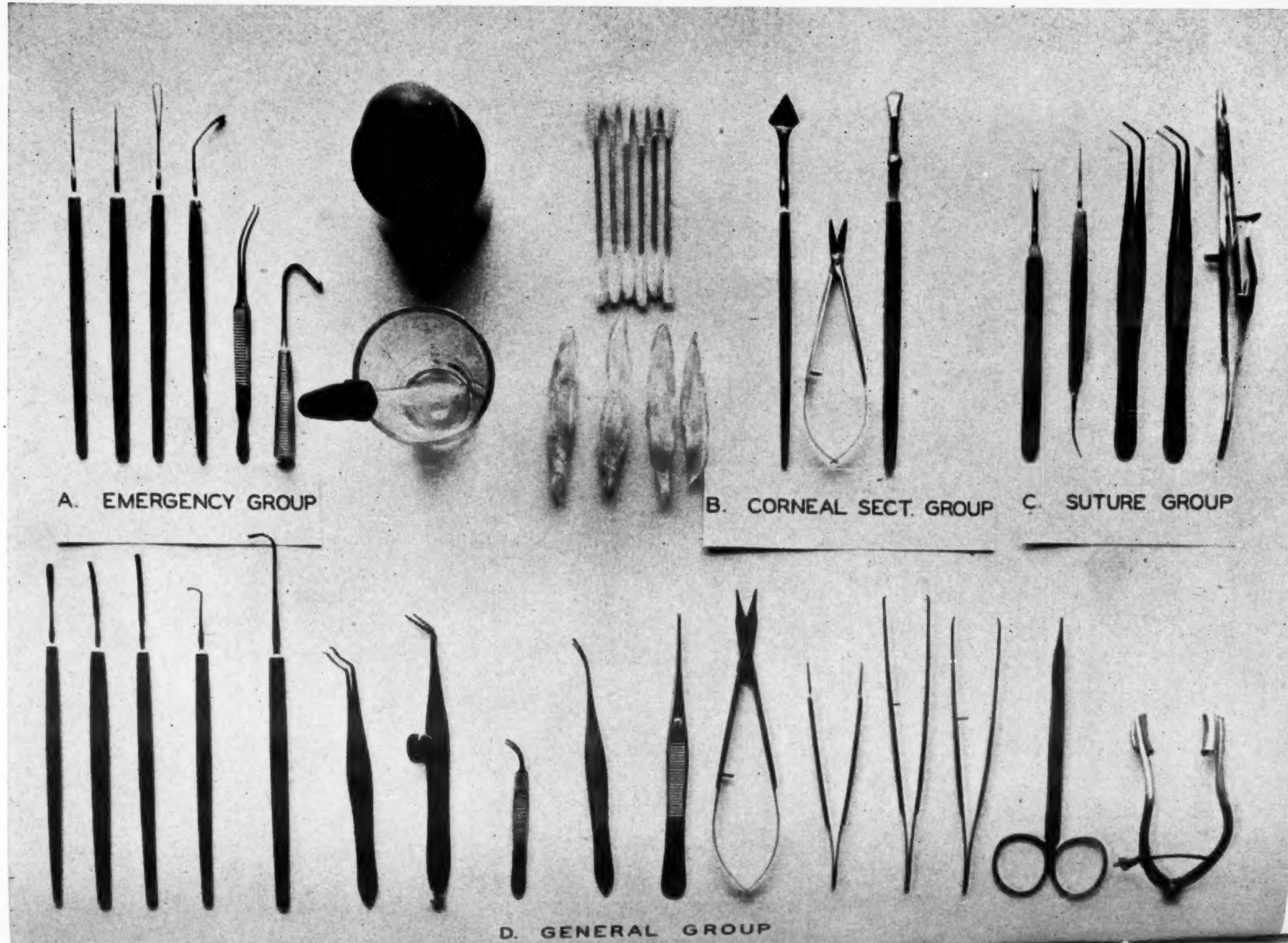
For local anesthesia, 4-percent cocaine is used. One drop is instilled four or five times, at three-minute intervals. If there is any hyperemia, a drop or two of 1:1,000 adrenalin is instilled after the first drop of cocaine.

SUBCONJUNCTIVAL INJECTION

After the third instillation of cocaine is completed, a subconjunctival injection of 0.2 c.c. of 1-percent cocaine, with 3 drops of adrenalin 1:10,000, is made above and below and well back from the cornea. This is massaged through the closed lids in order to diffuse it.

AKINESIA OF THE 7TH NERVE

In my cases the simple method described by O'Brien[1] of obtaining akinesia of the 7th nerve is routinely employed. Dr. Lancaster frequently does not paralyze the orbicularis, finding it unnecessary when his basal and local anesthesia are so well done. If one is not sure, he heartily


A. EMERGENCY GROUP
B. CORNEAL SECT. GROUP
C. SUTURE GROUP
D. GENERAL GROUP

recommends securing some degree of akinesia, first in importance being that of the extraocular muscles, and, secondly, the orbicularis.

About 3 c.c. of 2-percent novocaine, with 3 drops of 1:1,000 adrenalin added, are injected, using a 5 c.c. syringe and a 24-gauge, 1¼-inch needle.

RETROBULBAR INJECTION

I recommend the retrobulbar injection of 1 c.c. of 2-percent novocaine, with 3 drops of 1:1,000 adrenalin added, into the muscle cone in the region of the ciliary ganglion. This secures additional anesthesia and some degree of akinesia of the extraocular muscles, which, by their spontaneous contraction, are a too-frequent cause of vitreous loss.

An added, and frequently desired, effect of the retrobulbar injection of novocaine and adrenalin is the reduction of the intraocular pressure. The use of adrenalin increases the anesthetic effect of the novocaine as well as lessens the bleeding.

Too much stress cannot be laid on the importance of good basal and local anesthesia, and akinesia in cataract surgery. Adoption of Dr. Lancaster's routine gave me confidence from the very beginning in my own work at Dartmouth. Speed in ophthalmic surgery is not possible in a small general hospital where nurses unacquainted with ocular surgery may be in attendance. I found it was not necessary after following Dr. Lancaster's procedure in the use of anesthesia.

Instruments

Success in cataract surgery depends on the attention of the operator to a multitude of details rather than to any single procedure. Often the failure to attend to some single detail will lead to a disappointing result, although the surgeon may have selected the best procedure for the case.

I know of no operation wherein rhythm, precision, and coördination of the movements of both operator and assistant are so necessary. A skillful surgeon will manage to secure a good result in spite of handicaps. He does not rely upon his skill alone but makes certain ahead of time that he has good light, suitable glasses, and that he will not have to work in an awkward position nor with imperfect instruments.

It is well to give considerable attention to the selection and arrangement of the instruments on the instrument tray (fig. 1). The instrument tray is placed about three inches above the chest of the patient, in full view, where it is accessible to both myself and the assistant.

Group A is the emergency group; and these instruments are always placed in the upper left corner of the tray. Here they are out of the way; but when occasion for their use arises, I or the assistant knows immediately where to find them.

Group B includes the three cutting instruments employed in making the section.

Group C is the suture group.

The instruments in group D are arranged from right to left in the approximate order in which they are used as the operation progresses.

In addition to giving careful attention to the instruments employed in his cataract surgery, Dr. Lancaster has a sterile table set up in the operating room, upon which he has placed the various sterile solutions, syringes, medicine droppers, applicators, cotton, dressings, sterile sheets, surgeon's gloves, and the like (fig. 2). The adoption of this part of Dr. Lancaster's operating-room technique will prove of tremendous help to anyone doing ophthalmic surgery in a general hos-

Fig. 1 (Roper). Arrangement of instruments for cataract extraction.

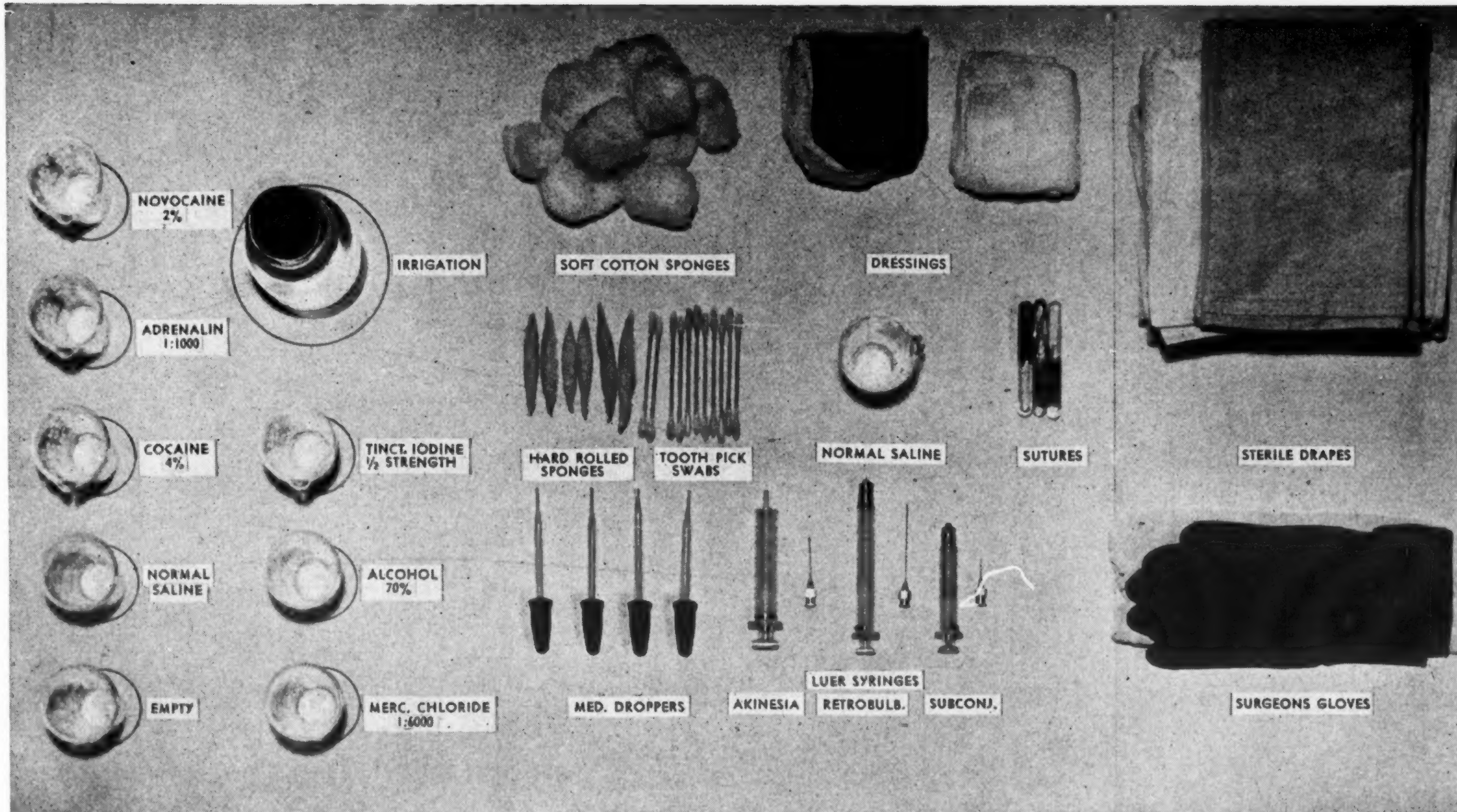


Fig. 2 (Roper). Sterile table for ophthalmic surgery.

pital where ophthalmic surgery constitutes a relatively small part of the work.

SUCCESSIVE STEPS OF THE OPERATION

Canthotomy. The practice of making a small canthotomy is advocated by Dr. Lancaster and should always be done when there is need for it.

Bridle suture. Good fixation is most important in cataract surgery. The eye in the orbit is fixed at only one point; namely, its own center of rotation. If rotation of the eye is to be prevented, fixation becomes necessary at two more points. The bridle suture serves as a point of fixation when held by an assistant, and both Dr. Lancaster and I employ it routinely.

Conjunctival flap. A small conjunctival flap is turned down on the limbus around the entire upper half of the eye. At the completion of the operation the entire wound is covered by conjunctiva, which affords added protection and better surgical closure.

Corneoscleral sutures. Dr. Lancaster advocates the use of three corneoscleral sutures. I always employ two and frequently a third. These are placed somewhat similar to a technique described by McLean.[2]

The sutures are placed in solid corneal and scleral tissue and not in loose, yielding conjunctiva. They are placed before the section is made, which eliminates the hazards of manipulating the opened eye. The sutures are so placed that they pass through and not over the lips of the wound. When sutures so placed are tied, the tissues are brought back to their exact preoperative position.

Gaping wounds are too frequently the cause of serious postoperative complications. Dr. Lancaster's method of suturing eliminates almost entirely the possibility of a gaping wound. The incidence of postoperative hyphema, iris prolapse, and high astigmatism is markedly reduced.

An incision or groove is made along the line of the contemplated section at the base of the conjunctival flap, about one half of the thickness toward the anterior chamber.

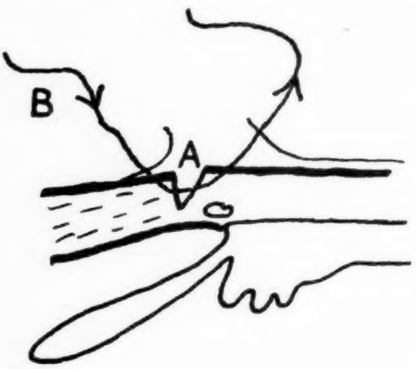
McLean uses a Lundsgaard knife in preparing his groove, stating also that a cataract knife, keratome, or small, sharp scalpel could be used for the same purpose. It is here that Dr. Lancaster's ingenuity has again come forward to help the ophthalmic surgeon. He has designed a guarded scleral knife (fig. 3) with which the incision part way through the sclera is made quickly, easily, and safely. It eliminates the unwelcome complication of inadvertently penetrating the anterior chamber. The guarded blade is set and the incision made obliquely, as it is made with a keratome. The operator can bear down firmly on the sclera and is able to complete the groove with a very few movements of the knife back and forth along the track.

Fig. 3 (Roper). Lancaster guarded scleral knife.

Good fixation is essential in preparing the groove. Dr. Lancaster uses a formidable looking "ice-tongs" forceps. Once he applies these to the globe they are not released until the groove is completed, sutures placed, and the anterior chamber opened. I have found this part of Dr. Lancaster's technique difficult because during all this period the left hand is fully occupied, which makes it necessary to depend on skilled assistance. The sharp points penetrate into the anterior cham-

ber, and I have noted the iris to bleed slightly, so that I am convinced the trauma might even be extended to the lens capsule. I have therefore adopted the use of a Gifford's fixation forceps with spring catch, with which the sclera laid bare by the conjunctival flap is grasped at points that will enable me to prepare short grooves and insert my sutures with a minimum of trauma to the tissues. To aid fixation I have the assistant grasp the later tied, will bring the lips of the wound back to their original position before the eye was opened. In addition, the edge of the loose conjunctiva above is brought down into position to meet the edge of the narrow strip of conjunctiva attached to the cornea (fig. 5).

Dr. Lancaster advises practicing passing the needles through leather, for instance a leather belt, to get the knack of doing it with the minimum of pressure.



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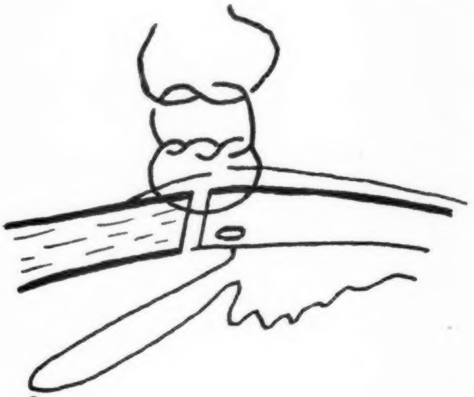

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Fig. 4 (Roper). Diagram showing A, primary incision by Lancaster guarded scleral knife; B, course of needle and thread passing through first the corneal and then the scleral lips of the groove, and finally through the edge of the loose conjunctiva above.

Fig. 5. Showing closure obtainable after extraction of the lens; lips of the wound back in their preoperative position and covered by conjunctiva.

It should be noted that the course of the suture differs from that described by McLean.[2]

bridle stitch, and while I am inserting the sutures he holds a pointed counter pressor against the sclera, which assists greatly in passing the needle through the corneoscleral tissues.

Special 8-0, double-armed silk sutures, with drum-tested needles, are used. The needle is started in clear cornea below the base of the conjunctival flap and run through both the corneal and scleral lips of the groove, emerging in scleral tissue about 1 to 1.5 mm. from the line of incision. The suture is then passed through the edge of the loose conjunctiva above (fig. 4). A suture so placed, when it is

Either a blunt or sharp Tyrrell hook is used to pull the suture out of the groove, and the loops are properly disposed of in preparation for making the section.

The operator should place his three sutures equidistant from each other at 10:30, 12, and 1:30 o'clock on the corneal dial. The suture in the vertical meridian at 12 o'clock is most accurately placed for obvious reasons. This suture I omit at times, and again I insert only the corneal portion in the beginning, leaving the placing of the scleral portion until after the extraction is completed. When this is

done, it is important to have a good forceps for picking up and holding the edge of the wound. The tissues should be grasped carefully and accurately, so that the needle can be passed close to the bite of the forceps. When the central suture is not passed through the scleral edge of the wound it can be used as a traction suture for elevation of the corneal flap, thus giving a direct view of the iris and the lens capsule.

One of the main objections to placing sutures after the section is made in positions on the corneal dial other than at 12 o'clock—which leads to malposition and buckling of the cornea later—is the tendency to run the sutures vertically instead of radially. Corneoscleral sutures of any kind that are not exactly appositional not only do not insure exact closure of the wound, but they prevent it and favor prolapse. By the Lancaster technique this danger is avoided when the sutures are placed before the section is made.

Completing the section. The anterior chamber is now entered above with an angular keratome. The section is completed with corneal scissors, care being taken not to cut the sutures. When the loops are held apart, one by the assistant and the other by the operator, it is comparatively easy to cut between them; the groove already prepared is made to gape (being spread) by slight traction on the opposing loops of the suture. The loops of the sutures are shortened at this time, the operator making certain that sufficient slack remains to allow ample gaping of the wound for the extraction. A surgeons' knot is placed in each lateral suture, and the ends laid out carefully so that they can be easily identified. These are not fully tied until after the extraction, and are reinforced by a third turn. Two pairs of Nugent's utility forceps are particularly suited to handling and tying the sutures.

The use of a binocular loupe considerably facilitates the placing of sutures and should be employed for the completion of the incision.

The extraction. Extraction in capsule is the operation of choice for the great majority of cases. For simplicity of technique and a minimum of trauma, removal with forceps is the method to be followed. Kalt forceps are used. When I feel that a capsulotomy is indicated to begin with, I make a deliberate one, not an accidental one, and make it with a suitable instrument. Chances should not be taken that might result in an accidental capsulotomy, which generally consists of an irregular tear running into the equatorial region and an accidental zonular tear as well. It is largely the condition of the zonule that will decide for or against extraction in capsule.

The closed forceps are introduced into the anterior chamber and are passed down to the lowest point possible, but should avoid any portion of the iris when they are opened. The forceps are now opened and pressed gently against the lens to grasp the capsule and, at the same time counterpressure is applied at the limbus below with the heel of a strabismus hook. The counterpressure is then continued by slowly moving the hook along the limbus between the 4- and 8-o'clock position. No traction is made on the inside until the zonule is broken below and the iris is seen to be raised by the lens as it begins to tumble. From this point it is my opinion that the larger percentage of successes lies in the skill with which the counterpressure on the outside is coordinated with the very slight but simultaneous traction on the inside.

The assistant is instructed to stand ready to use the heel of a small Stevens

muscle hook to ease the edge of the iris over the lens should I so signify; or with a hook, if it is an extracapsular extraction, to ease out the lens by a rotary movement as it presents itself in the wound.

When the lens is thus removed by tumbling it out of its seat on the face of the vitreous, and the iris is seen to fall back into the patellar fossa, with the pupil round and fairly well contracted in this position, it is a pleasing sight to the operator.

The iridectomy. Dr. Lancaster prefers a basal buttonhole iridectomy, and I use it in all cases except in instances wherein the pupil is not large enough to permit the passage of the lens. This requires that the sphincter of the iris be cut, but usually the pupil is dilated sufficiently by the euphthalmine and subconjunctival injection of cocaine and adrenalin.

The purpose of the iridectomy is to establish a communication between the posterior and anterior chamber at the periphery, so that if there is any escape of aqueous from the anterior chamber through the wound it will not carry the base of the iris with it in a prolapse, even though it makes an escape with a gush. Many believe that the regular iridectomy is the best guarantee against prolapse and affords, at the same time, a large pupil for the passage of the lens. Dr. Lancaster has long held the opinion that a basal iridectomy that leaves an intact sphincter is a better preventive of prolapse.

The basal or peripheral iridectomy is best performed by having the assistant lift the corneal flap with the central suture. One gets a direct view of the iris and a limbus shelf if it exists. The point of the iris picked up should be pulled toward the center of the pupil, at the same time lifting it slightly and then cutting close behind the point where the forceps holds the iris.

Peripheral iridotomy can be practiced and is claimed to be as satisfactory as peripheral iridectomy.

Dr. Lancaster routinely performs his iridectomy after the extraction of the lens, claiming that the buttonhole is worse than useless as an aid to the passage of the lens, since there is always the danger that the lens might catch in the buttonhole instead of taking the proper route through the pupil. I have always felt more at ease performing the iridectomy before making the extraction, feeling that there was less chance of injury to the vitreous.

Completing the operation. After it is ascertained that the iris is not caught between the lips of the wound, the two lateral appositional sutures are drawn taut, tied with a surgeon's knot, and reinforced by a third turn. Loosely tied sutures are ineffective and favor prolapse. If irrigation or any extensive toilet of the wound is necessary, the lateral sutures may still first be tied, making these maneuvers much safer. A little massage over the cornea with a Daviel's lens scoop aids the tissues in returning to their normal relations. If the central suture has not been passed through the scleral edge of the wound, it is done now.

Before the eye is closed, two drops of 1-percent eserine are instilled, followed by 1-percent eserine ointment. This will draw the iris into the chamber and keep it from being incarcerated in the lips of the incision. Five-percent sulfathiazole ointment is added and spread across the closed palpebral fissure, covering the lashes well. At the time of the first dressing, 48 hours later, atropine may or may not be employed, depending upon conditions as they are found. Even if the extraction has been extracapsular, Dr. Lancaster does not put in atropine until the time of the first dressing, and then only if it is indicated.

A bilateral Barraquer dressing is applied. This consists of two or three layers of cotton laid over the lids and moistened down with sterile saline solution. When applied thus, the dressing approximates closely the curves and depressions of the lids. Over this is placed the regular bilateral eye dressing made of cotton and gauze, which fills in the two orbits with a nicely rounded convex dome. When such a dressing dries, the lids are very satisfactorily immobilized. A stiff Ring mask further protects the eyes from blows or the patient's impulsive thrusts. anxiety and apprehension. Ten grains of aspirin alone, or combined with a grain of nembutal, is generally given as soon as the patient is returned to his room. From 0.5 to 1 grain of codeine can be given hypodermically if necessary. Dr. Lancaster insists that his patient be made comfortable after operation, feeling that a restless patient is more apt to do harm to his eye than a comfortable one. In order to give relief to the patient's back, he has the head of the bed elevated from time to time so that the patient is almost in a sitting position.

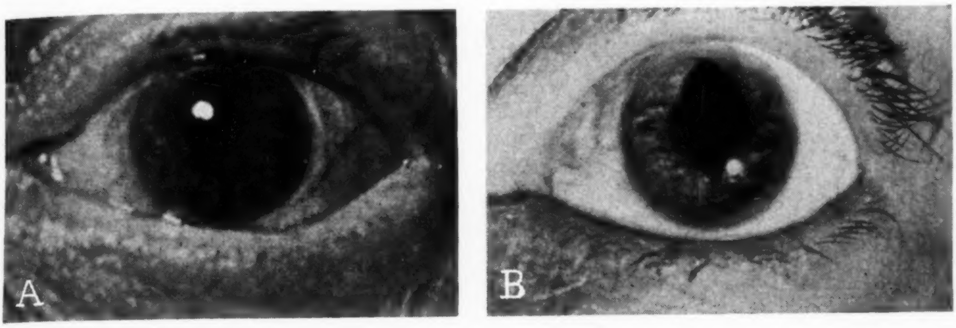

Fig. 6 (Roper). Appearance of eyes following intracapsular extraction of the cataract. A, round pupil with peripheral iridectomy; B, central iridectomy (sphincterectomy).

Comparative quiet in the operating room should be the rule. A patient may be calm and placid from his medication but he should not be disturbed by irrelevant conversation, particularly remarks about the operation or comments on defective materials which the careless surgeon thinks the patient is too sleepy to hear. Patients are keenly alert to comments by the physician. Admonitions to the patient have little value. Encouraging remarks, forebearance, and patience will place the physician in better command of the situation throughout.

Postoperative pain in cataract surgery is actually very slight, but careful consideration should be given to the patient's

It is my practice to remove the sutures between the tenth and twelfth days. A word about the removal of sutures is important. One must make certain of sufficient anesthesia for their removal. A small drop of 4-percent cocaine is instilled and, after a three-minute interval, one or two drops of 1:1,000 adrenalin. Two or three instillations of 4-percent cocaine should follow at three-minute intervals before an attempt is made to remove the sutures. The upper lid is then held up gently by means of a lid retractor held by an assistant. A good light, binocular loupe, sharp pointed scissors, and a Nugent utility forceps are other necessities.

Report of cases

The cataract extractions reported here are those done by me between November, 1941, and November, 1942, when Dr. Lancaster left the Dartmouth Eye Institute to resume practice in Boston. They constitute a comparatively small number of cases but are a series in which all operations were performed by me with a newly acquired technique taught me by Dr. Lancaster.

In all, 27 extractions were performed; 22 were of the ordinary senile type of cataract and 5 were complicated, of various types. Intracapsular extractions were attempted in all, with extracapsular extractions resulting in only four. In these four cases the capsule ruptured during the expression of the lens. The chief cause of rupture of the capsule appeared to be a contracted pupil, leading to faulty pressure with the hook and unnecessary traction with the forceps. There was one hemorrhage into the anterior chamber in the case of a woman who had hypertension and diabetes. There was one prolapse and one incarceration of the iris in the series. The average cylinder was +1.53D.

A second series might fairly be expected to give better results since these cases are the first done by this method.

Figure 6 shows two of the eyes in the series after operation.

Summary and Conclusions

Dr. Walter B. Lancaster's technique of cataract extraction is described in detail. The importance of careful preliminary treatment and good anesthesia is stressed. Successive steps of his operation are described, the most important being his method of placing corneoscleral sutures before the section is made.

Results in a series of 27 cases are reported wherein the author followed Dr. Lancaster's technique exclusively.

The high percentage of intracapsular extractions obtained, the low incidence of complications during and following surgery, and the low amount of astigmatism which resulted all seem to prove the merit of Dr. Lancaster's technique.

The lettering on the illustrations was done by Mr. Ranald C. Hill of the Dartmouth Eye Institute.

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CLEARING OF EDEMATOUS CORNEAS BY GLYCERINE

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Hypertonic solutions of glucose[1] and of sodium chloride[2] have been used as aids to ophthalmoscopy in the reduction of corneal edema. In the present communication I wish to recommend the use of full-strength glycerine for this purpose. Glycerine is more satisfactory in that it can be used simply in drop form, replacing the eye cup; it causes less smarting; and it clears the cornea more promptly. One or two drops are instilled on the upper portion of the unanesthetized cornea. The eyelids are held open, and the viscid glycerine is allowed to flow over the whole surface of the cornea. Clearing occurs in 20 to 30 seconds and lasts several minutes.

The use of glycerine as a clearing agent is indicated whenever clouding of the cornea is due to simple epithelial edema. It is suitable in most cases of steamy cornea with acute glaucoma, of idiopathic corneal edema, and the early stages of guttate cornea (Fuchs's epithelial dystrophy). When the edema has gone on to the formation of gross bullae, glycerine will have only slight effect. In this instance, it will clear the edematous portion of the cornea but will not evacuate the bullae. Glycerine has no appreciable effect on the stromal opacity, resulting either from stretching, as in some cases of glaucoma, or from increased stromal hydration, as in interstitial keratitis and following removal of the endothelium. Glycerine has, of course, no effect on the opacity of leucomatous corneas.

In our hands glycerine has been especially useful in examining patients who have come to the Clinic for the first time with acute attacks of glaucoma and in whom the steaminess of the cornea has prevented adequate examination of the eye. Glycerine is instilled, and the patient is examined with the ophthalmoscope and slitlamp microscope as soon as possible, since the clearing effect usually lasts less than five minutes.* If the cornea again becomes cloudy, glycerine drops may be instilled several times. I have noted no toxic manifestations of the glycerine in the 50 or more cases in which I have used it.

Since the effects of glycerine are so transient, the clearing might be thought to result from the formation of an optically homogeneous layer on the surface of the edematous epithelium. This is, however, not the impression one obtains on watching the clearing process; the steaminess does not begin to disappear for 15 to 20 seconds after the glycerine covers the cornea, and the clearing may often be seen taking place beneath the glycerine layer. The effect, like that from other hypertonic fluids, is therefore presumably due to removal of excess water from the hydropic epithelium.

The intraocular pressure is not affected by the glycerine used as outlined above, and no permanent beneficial effect in the cornea has been obtained from its repeated use. It is recommended merely as a simple diagnostic aid.

243 Charles Street.

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* The clearing effect lasts longer if glycerine is used in tablet rather than in liquid form. Glycerine tablets may be obtained by cutting up infant suppositories into appropriate sizes. These are then implanted in the lower cul-de-sac.

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OBJECTIVES OF ORTHOPTIC EXAMINATION AND TREATMENT*

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Orthoptics was rendered a poor service by those early enthusiasts who defined it the nonoperative, as opposed to the operative treatment of squint. There is but one way of treating squint; namely, by the purposeful integration of a number of different procedures, of which surgery, while the most dramatic, is but one, and orthoptics is another. To be successfully conducted, however, so many of the specialized aptitudes and attitudes for teaching and for reëducating are required of the practicing orthoptist, that this skill has emerged almost as a specialty of its own. For this reason, and for this reason alone, it can and should be treated separately, apart from the operative and other nonoperative procedures in the treatment of motor anomalies of the eyes.

I am purposely emphasizing the aspect of teaching in connection with orthoptics which, I believe, is a specialized form of education, of rehabilitation, a branch of what the Germans call "Heilpädagogik" as applied to individuals with faulty sensorimotor visual habits; or, in the terminology of Chavasse[1] and Lancaster,[2] with faulty visual "reflexes." Both its possibilities and limitations are those of learning and habit formation in general. Its possibilities, like those of all learning, are greatest in childhood, because of the child's greater pliability, but show another peak in the adolescent and the young adult, due to the greater incentive of motivation at that age.† Its limitations, again like those of all learning—apart, naturally, from the anatomic factors present in every individual case—lie in the individual's developmental past, in environmental factors, and in differences of motivation. Given equal sensorimotor ocular conditions, treatment will be less successful if, for example, a child or its parent is dull, or, to cite another example, if a child's family is of lower social standing and is repressed rather than driven by its social inferiority; it will be more difficult, if, for example, the parents are divorced. I have no statistics to offer, but have seen squint in its repulsive convergent form so often in, for example, the older of two boys of one family, but none in the younger, that I feel that environment, emotions, and what the psychoanalysts call psychic trauma are at least as important in the etiology of squint as heredity. Certain forms of squint have been called "purposive," the squint's purpose being somewhat like the avoidance of diplopia. It seems to me that squint may be "purposive" also in quite another sense; namely, in the sense in which many other manifestations of disease may prove to be purposive in a neurotic child.

This paper deals with the objectives of orthoptic examination, and it might therefore seem that such remarks are beside the point. I believe, however, that to ac-

* From the Dartmouth Eye Institute, Dartmouth Medical School. Read before the New York Society for Clinical Ophthalmology, January 4, 1943.

† Orthoptists differ as to the most advantageous age for orthoptic treatment. I had the best results with college students, simply because this is the group of people to which I feel most attached and which I can handle best. Cantonnet and Filliozat,[3] who stress so much the "mental effort" in orthoptic reëducation, naturally also prefer somewhat older patients and claim best results with them. When most orthoptists, especially women, emphasize that young children show best results, it means that they are at their best with young children. Success in orthoptics, as in teaching in general, is, to a great extent, a matter of personality.

quire as much insight as possible into the past history and present circumstances of the squint is a part of this examination. Every bit of information relating to the background of a case, even though nonvisual in character, will help in arriving at a general idea of what may be expected, and in deciding which of many possible steps should be taken in the individual case.

What, then, is an orthoptic examination? Properly speaking, the ophthalmologist's entire examination of a patient with sensorimotor visual disturbances is an orthoptic examination, just as the prescription of a proper refractive or aniseikonic correction, of prisms, of monocular occlusion, and the like, is part of the orthoptic treatment. One is accustomed, however, to designate by orthoptic examination, either simply the use of orthoptic instruments for diagnostic purposes, or that part of the examination only which is concerned with the following two points: (1) the determination of a patient's mode of fixation and the measurement of the deviation, if any, of his eyes from that position in which bifoveal fixation can or should be maintained; and (2) the determination of the binocular visual habits heretofore acquired by the patient.

I shall have to explain the somewhat complicated wording of these two points. Moreover, I admit that the classification into these two points is, like most systematizing, somewhat artificial. It would have been simpler to have designated the first step of the orthoptic examination in the usual way, as the determination of the position of the eyes. The orthoptist, however, determines not a position of the eyes, but the line of regard of one eye as referred to the line of regard of the other eye, or, in other words, the behavior of one eye while the other eye is fixating. Thus, this measurement already takes into account two of the very basic perceptual habits (or "reflexes," if one prefers); namely, the habit of fixation and the habit of selective attention. While determining what one calls the position of the eyes, one is already dealing with the visual habits of the patient.

Briefly, point (1) is the measurement of the patient's phoria or tropia, which should be taken at infinity (that is, with relaxed accommodation) as well as at some near point, say 40 cm. (that is, with induced accommodation of 2.50D.).

So many devices are used to determine phoria and tropia that one usually forgets to look at the patient without "embarrassing" him (Chavasse[1]) with any of the instruments. But if one allows the patient to look naturally at an object straight ahead (a light is best), at least 15 feet away, one gets a good idea of the way he usually fixates simply by observing him. One may even get a first lead to a diagnosis by noticing whether or not the patient's head is tilted (vertical imbalance), his chin is lowered toward the chest (esophoria), or elevated (exophoria), and so forth. By covering one of the patient's eyes for a split second, a time too short for the dissociation of the eyes, one may get at least an idea as to whether or not the patient has bifoveal fixation (Burian[4]).

The use of the Maddox rod, of the red glass of Bielschowsky, or of the cover test (also known as screen and prisms, or screen and parallax test) is familiar to all. How to use any simple stereoscope for the determination of the angle of deviation, especially in phoria, has been described by me in a previous paper.[5]

The determination of the amount and direction of a phoria or tropia with the synotophore (or any of the major amblyoscopes) is a simple matter. The advantage of these instruments is that they can be adjusted for measurement of the

vertical, as well as of the horizontal deviations and for any cyclophoria that the patient may have. Some fixation target must be presented alternately to each eye and the arms of the instrument adjusted until there is no movement of either eye on alternating flashes. Of course, in order to make this test valid, one must ascertain beforehand that the patient is able to fixate with either fovea, and that he does so alternately. If the patient fixates extrafoveally with one eye (as may be the case in high amblyopia of that eye), the examiner must rely on the position of the corneal reflex produced by the lamp of the instrument. That such a reading may be less accurate, due to the so-called angle gamma, is merely mentioned in passing. The deviation, if any, from orthophoria can be read off the scales provided separately for the horizontal, the vertical, and the cyclo-deviations.

The optics of the major amblyoscopes is such as to necessitate no accommodation for focusing the targets. The values arrived at are, therefore, an indication of the position of the patient's eyes when focused for infinity. For measurements at near, say 40 cm., lenses of -2.50D. sph. have to be put into the lens holders provided for this purpose. These lenses will induce an accommodation of 2.50D. for proper focusing. Theoretically, 2.50D. of accommodation goes hand in hand with 7.50^{Δ} of convergence in each eye (if the pupillary distance is 60 mm.). In orthophoria for near, the patient therefore will set each of the arms of the instrument at 7.50^{Δ} "base-out." Measurements of phorias or tropias have to be based on these points as zero readings.*

* In some instruments—for example in the stereo-orthoptor—the optics is so arranged that plus lenses have to be added for distance measurements, and removed for near. Otherwise the principle is the same.

Much more important, and, of course, much less simple is what was mentioned above as point (2); namely, the analysis of the patient's binocular behavior, his binocular seeing habits.

Let me emphasize again the words *binocular* habits. It is erroneous to state, although this is frequently done, that a squinter uses only one eye. Vision is always a binocular function, even when one eye is amblyopic, even when it is occluded. Stimuli originating in the squinting eye will produce waves of neural activity in the cortex which, on their part, interact with, modify, facilitate, or inhibit neural activity originating from stimuli of the other eye. Thus the squinter has binocular vision; he has developed binocular habits of his own. His habits, to be sure, are different from those which we call normal: they are anomalous binocular visual habits.

"Fusion" of two uniocular patterns of stimulations is the most characteristic, the most outstanding of the normal binocular visual reflexes, habits, or responses (whichever term one prefers). The examination by the orthoptist for binocular habits has to start with tests for the presence or absence of fusion.

With the synoptophore, fusion is tested by the examiner setting the arms of the instrument at the patient's objective angle of phoria or squint and presenting two targets of patterns sufficiently similar to be fused.

The classic pattern for the determination of fusion is the well-known F-L-Chart. When fused, the patient reads it as an E. If one or the other eye should not participate in foveal perception, F or L would be read, respectively. This chart gives all the important features of a good fusion target in the simplest form. What is needed first is an incentive to fusion; that is, parts of the two patterns must be identical. In the F-L-Chart, it is the

vertical bar that is common to the two patterns and is fused. Next, the two patterns must present some uniocularly visible parts to indicate to the examiner whether both eyes are coöperating in the perception; the horizontal bars of the chart serve this purpose. Moreover, each of the respective uniocular patterns must be meaningful in itself ("F" or "L") and no suggestion of the binocular pattern ("E") be given in either of the patterns perceived uniocularly.

If a pattern like the F-L-Chart is unified at least at the objective angle of squint, the ability of the patient to fuse has been established whether or not he avails himself of this faculty under ordinary circumstances.

For testing the normality or anomaly of sensory correspondence—the next step of an orthoptic examination—the best patterns are the classic ones designed by Miss Maddox and her followers: the soldier and the sentry-box, the lion and the cage, the clock and the hands, and the like. These targets are to be preferred to any such targets as the aforementioned F-L-Chart, because, in contradistinction to the latter, they provide no incentive to fusion. The soldier, presented to one eye, must be put by the patient into the sentry-box, seen by the other eye, by moving one or both arms of the synoptophore. If the patient is able to perform this task—that is, if he is able to put the arms of the instrument in a position in which the soldier appears to be in the house—he has indicated that, at that particular setting of the instrument he sees the two patterns (each of which is seen by one eye only) in one and the same direction. Selective attention being directed to the patterns, at least one of them will be seen in foveal fixation, while the other pattern will be directed toward that area of the retina which habitually shares selective attention with the fixating fovea. If the area selected in the second eye is also the fovea of this eye—that is, if it is the two foveas that are habitually selected in unitary acts of attention—the patient's sensory correspondence is normal and corresponding retinal points of *his* eyes, especially his two foveas, *have* common visual direction.

This is, of course, not always the case in squint. In the retina of the squinting eye an area other than its fovea may assume, through learning, the role of being turned, with the fovea of the other eye, toward a visual stimulus when the reflex of selective attention and the fixation reflex react to this stimulus. This retinal area in the squinting eye is what is called its "false macula." The result of the habitual selection of these two areas in unitary acts of attention is again commonness of their respective visual directions, while any object presented to the fovea of the squinting eye will be perceived as being in a secondary, a different visual direction. This is that anomalous binocular visual habit of the squinter which is best called "anomalous sensory correspondence."

In the synoptophore, patients with "anomalous sensory correspondence" will place one of the patterns in line with the fovea of the fixating eye and will at least attempt to place the other pattern in line with the pseudofovea; if this is accomplished, the patterns will be experienced as being in the same, in a common visual direction. Patterns aligned with both "true" foveas, on the other hand, will be perceived—so long as one of them is not constantly suppressed—as being in two different directions.

Anomalous sensory correspondence cannot be tested in the stereoscope. It is difficult to test it in the stereo-orthoptor.

The classic way to determine the presence or absence of anomalous sensory correspondence is by Tschermak's afterimage

test, repeatedly described by Bielschowsky[6] and recently again called to our attention by Lancaster.[2] These descriptions are easily accessible, and I shall not repeat them. The other test used and described by Bielschowsky, his red-glass test, also needs merely to be mentioned for the sake of completeness. The behavior of people with no established sensory correspondence is especially characteristic in these two tests.

The next step in the orthoptic examination of patients who had heretofore shown normality of their binocular visual habits is concerned with the adaptability of the appropriate sensorimotor reactions which constitute fusion, called "fusional amplitude" in clinical language.*

Amplitude of fusion is a response based primarily on "usage." Large amplitudes are not always the most desired; moreover, they may indicate that fusion operates under additional stress. A person who, for example, overcomes 6 or 8 or 10 prism diopters vertically in one direction, without breaking fusion, certainly has a latent paresis of a vertical motor which may have become totally concomitant. Such a habit of overcoming obstacles to fusion may or may not be desirable. It may be the cause of so-called eyestrain. It may break down under trying conditions. For a combat plane, I would rather prefer a pilot with 2 or 3 prism diopters of vertical fusional amplitudes, equally distributed.

The fundamental discovery that the amplitude of fusion can be enhanced by training was made by Hofmann and Bielschowsky[7] some 40 years ago. Since then, fusion training has become the most successful branch of orthoptic treatment. Esophoria, convergence insufficiency, exophoria with and without spasm of accommodation, and periodic divergent squint are among the conditions in which orthoptic treatment is chiefly indicated. This is done most conveniently with one of the major amblyoscopes, with the rotoscope, or the orthoptor. My paper[5] may be consulted as to how the stereoscope may be used for this purpose.

I shall not here deal with the technique of fusion training, and shall also omit discussion of the next step of the orthoptic examination and the most conspicuous of visual responses in the normal individual; namely, depth perception. Even to scratch the surface of this most controversial chapter would run beyond the scope of this paper. Depth perception in the synoptophore or any other orthoptic instrument has to be tested with stereograms; that is, two similar, but disparate patterns prepared according to the principle discovered by Wheatstone more than 100 years ago. Various graded stereograms have been developed lately, with the aid of which a numerical evaluation of the faculty of depth perception is possible.

It has been claimed repeatedly that patients with anomalous sensory correspondence develop not only fusion, but also some depth perception. I have never seen such a case.

What, then, is the orthoptist's objective in the treatment of squint? It can be given in one word; namely, *reëducation.* A faulty habit, a faulty adaptive reaction has to be broken up and a new one developed. I wish to stress the words, adaptive reaction. The squinter's binocular

* The two great authorities on oculomotor nomenclature, Chavasse and Lancaster, propose the term "vergence" for fusion movements, which is certainly the best designation. In this connection, I shall use the term "fusional amplitude" or "amplitude of fusion" simply because it is not only more familiar, but stresses more the habit character of these sensorimotor reactions. The term "ductions," so often used, is definitely a misnomer. "Duction," as both Chavasse and Lancaster have stressed, is that which is performed by *one* eye only, regardless of the performance of the other eye. It is a term of uniocular behavior.

visual habits are faulty, are anomalous only when compared with the habits of those whom we call normal. But, in their way, the squinter's habits also have adaptive value and his spatial localization, if not correct, is at least corrected for everyday use.*

This being the case, is orthoptic training of the squinter of any value? It certainly is. Normality is always the goal if and when it can be attained.

I shall say only a few words about the orthoptic training of squinters. It is its objectives, not its technique, that I have undertaken to discuss. As already mentioned, the breaking of an established faulty habit is the first step to be taken. Accommodative convergent squint will be used as an example. The squint being due to excessive convergence associated with accommodation, full correction has to be effected first. If the sensory correspondence is normal and fusion can be attained by glasses, the fusional amplitude, the vergence, and, first of all, the dissociation of accommodation from convergence have to be trained. This can easily be done on the synoptophore; or with any instrument like the rotoscope, the stereo-orthoptor, or the telebinocular. How it can be done with the use of a simple stereoscope has been reported.[5]

It is a matter of opinion whether or not accommodative squint can be called "cured" if glasses are necessary to keep the patient's eyes straight. In a number of publications, Davis[8] has stressed the fact that accommodative squinters should be trained to dissociate accommodation and convergence *and* to keep their eyes straight without glasses. We have recently had a student at Dartmouth College, who had been wearing glasses for a number of years, who could be trained not only to regain normal sensory correspondence, to develop simultaneous perception, fusional amplitude, and a sufficiently good evaluation of depth, but was also taught how to discard his glasses for at least a part of the day without relapsing into his faulty convergence habits.

Nonaccommodative squint is, first of all, a surgical problem, and orthoptic training has to be timed with reference to the surgical procedure. The best way to break the habit of anomalous sensory correspondence, the habit of turning a false macula upon the object of regard, is by surgery. Even continuous occlusion is, in itself, unsatisfactory. If the angle of squint measures but a few degrees, prisms, constantly worn, help to condition the two foveas to the same stimulus.

As to the so-called retinal "massage" in the treatment of anomalous sensory correspondence, I think its value lies in the reconditioning of attention. Nothing "catches" attention so easily as flashing lights or motion. As soon as the patient is able to catch the two patterns in *one* act of attention, they will appear in one and the same direction.

Most orthoptists use the same patterns for the treatment of anomalous sensory correspondence and for its diagnosis. I do not agree with this usage. I believe greatly in interaction, summation, fusion between similar patterns of brain activity; therefore I prefer fusion patterns such as present similar stimuli to the two eyes to patterns of the lion-in-the-cage variety, for this purpose. The action of the former is twofold. They stimulate fusion and thereby reinforce the habit called "normal sensory correspondence."

Summarizing, I wish to anticipate one of the most frequent questions that are asked of one taking a stand for orthoptics; namely, why is there no consensus

* As contrasted with the confusion and diplopia of a person with recent paralytic squint, a squinter thus adapted will see things as they are and, with certain limitations, where they are.

of opinion as to its value? Immediately, two great names come to mind. Bielschowsky did not think too highly of orthoptics; he believed in centers not in habits. Nor did Chavasse have much use for it.

Chavasse was so much under the spell of the great Russian reflexologists, Pavlov and Bekhterev, as to believe with them that reflex pathways are something anatomically defined, in the way one is accustomed to seeing them depicted in a textbook: he believed that all those "conditioned" visual reflexes so aptly described by him were representations of so many new, definite connections, unchangeable once they achieved what he called "unconditioned fixity." That is why he did not believe in orthoptics.

This is not the time, and I certainly lack the competence, to discuss the pros and cons of this trend of "localizing" in science and in medicine. Our thinking is still too much in terms of cells, pathways, and centers to be free for the full appreciation of function. There are leading minds in medicine and psychology, such as Goldstein, Köhler, Lashley, Collier, who dealt with this question and may be consulted by anyone who is interested. One must have the functional approach to habit and to performance in order to believe in orthoptics.

When asked: Do you believe in orthoptics? I cannot help answering with a question: Do you believe in learning?

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WALTER B. LANCASTER, AN APPRECIATION

The last hundred years have witnessed wonderful achievements in medicine, fully comparable to those in science, of which not the least have been in our own field of ophthalmology. Many names have found a place on the mythical roll of honor. These have been written there for original contributions, for teaching, for caring well for many patients, or sometimes for two or even all three of these qualifications.

There are enshrined in the minds of all of us names of men whom we revere for what they did and what they were, a different list for each of us, no doubt, depending on our contacts and on those whom our teachers eulogized. Older ophthalmologists who studied in the clinics of Europe think especially of such men as Von Graefe, Donders, Snellen, Fuchs, Axenfeld, Nettleship, Elschnig, Bowman, de Lapersonne, Morax, Gonin, de Grosz, and many others. They recall the original contributions of these men and their great teaching clinics.

In the United States the first important names were naturally in the East, then followed those in the Middle West, and already one may guess at names from the Far West that will find a place on the honor roll. New York has given us Agnew, Noyes, Callan, Gruening, Duane, Wheeler; Philadelphia: de Schweinitz, Harlan, Risley, Sweet, Holloway; Bos-

ton: the Derbys, Williams, Standish, Howe, Greenwood. When we think of Buffalo, Park Lewis comes to mind; Baltimore contributed Theobold and Woods; Washington, Wilmer; Cincinnati, Sattler and Vail; Chicago, Wilder, Wescott, Gradle; New Orleans, Feingold; Saint Louis, Green, Ewing, Alt; Omaha, Gifford; Denver, Jackson. It is not difficult to predict at least some of those now living who will be honored in the future for their accomplishments of the present.

The purpose of this issue of the Journal is to honor such a man. In our pioneer years Massachusetts was the leader in business and politics. And for many years after its political importance waned, it still remained, with Boston as its hub, the intellectual center. In this background Dr. Walter B. Lancaster has spent most of his medical life, and all who have come in contact with him must have felt that he well exemplified this culture. For long he has been in the fore in the three great contributions that the professional man can make: research, teaching, and clinical practice. He has the original type of mind, as witness his many articles in this and other scientific journals pertaining to new ideas in surgery and therapeutics. I was relatively young in the practice of medicine when on one occasion he visited Saint Louis and gave a surgical demonstration. I have true respect for anyone who can do this well. So many try unsuccessfully. There are such a multitude of unforeseen difficulties, strange assistants so officious as to end by hindering, unsatisfactory patients—generally the ones that have been put off by the home doctor because they were too poor risks—unaccustomed instruments, and a very critical and too intimate gallery. Dr. Lancaster was quite unruffled, deliberate, accepting the situation philosophically and without criticism, and proceeding to perform the expected operations with great skill. I then first saw his method of injecting the extraocular muscles to prevent their contraction during cataract extraction, and heard him expound his thoughts on the matter. He gave us much food for consideration on that day.

To mention only a few of his other contributions of value one might cite his magnet devised to produce the maximum pull for its weight; his astigmatic dial; his charts and methods of plotting diplopia fields; all of which are now employed in our own clinic. He was one of the first to recognize the importance of aniseikonia in eye strain and to prescribe lenses for its correction.

For many years he has been a leader among ophthalmologic teachers. His clear expositions of the action of the external muscles and of the abstruse subject of physiologic optics will be remembered by all of his students.

It is difficult to evaluate the extent of the direct clinical contributions of a physician, but, though intangible and immeasurable, perhaps these are the most important benefactions of his life, and certainly for many years Dr. Lancaster has done more than his share in this field.

It is with sincere good wishes that the Journal extends greetings to Dr. Lancaster on his eightieth birthday, and hopes that he may live long and continue to be the same unfailing source for good in his profession and to give happiness to his many friends in the future as he has in the past.

Lawrence T. Post.

PAN-AMERICAN NOTES

Edited by Dr. M. Uribe Troncoso
500 West End Avenue, New York

Communications should reach the editor by the twelfth of the month

Miscellaneous

Latin-American developments in the prevention of blindness. Dr. Moacyr E. Alvaro of Brazil, in a report published by the National Society for the Prevention of Blindness, states that active efforts for protection of eyesight and the conservation of vision are now under way in many of the Central and South American countries through legislation and public education along the lines which have been successful in the United States.

"In the last fifteen or twenty years," the report states, "a number of committees for the prevention of blindness have been formed in the various Latin-American countries. The great majority of these groups have in one way or another established bonds of friendship with the National Society for the Prevention of Blindness in the United States, and an active correspondences enables an exchange of views which have proved very beneficial; the older sister has provided the younger sisters with much useful advice.

"Some of these agencies for prevention of blindness in South America have brought about the establishment of routine examination of the eyes of school children, measures for the protection of workers from accidents in industry, better medical care for eye patients, and legislation to make compulsory the use of a prophylactic solution in the eye of infants at birth as a safeguard against ophthalmia neonatorum.

"Substantial progress has been made or is being made in campaigns against syphilitic, gonococcal and tuberculosis infections, in general measures against communicable diseases, better industrial hygiene, multiplication of local health agencies, improvement of housing conditions and nutrition habits, better facilities for the care of the sick, higher educational standards, etc.

"The data now being collected by the Committee for the Prevention of Blindness of the Pan-American Congress of Ophthalmology will prove of great use in finding out what the local problems are in each region, so that adequate steps may be taken in order to solve them specifically.

"A great problem in Latin America is the fact that there are only 1,000 ophthalmologists scattered over the whole area to give their services to a population of 120,000,000 people. This number, small in itself, is relatively still smaller when the difficulties in transportation have to be taken into consideration.

"Both the translation and adaptation of text books used in the United States into Spanish and Portuguese with the aid of professors of ophthalmology of the Latin-American countries and the increasing cultural exchange between ophthalmologists north and south of the Rio Grande, will aid in improvement of the facilities for treating eye cases."

Asociacion para Evitar la Ceguera en Mexico (Association for the Prevention of Blindness in Mexico) will celebrate its twenty-fifth anniversary with a scientific week from the 13th to the 21st of the coming month of August. It invites contributions to its program from ophthalmologists in the United States, to be presented and discussed at the sessions. In order to provide sufficient time for arranging the contributions which will appear on the program, authors' works or their titles should be sent before July 15, 1943, to the address of the Association, Gómez Farías 19, Mexico City. The notice of the meeting is signed by Dr. Manuel Márquez, Dr. Luis Sanchez Bulnes, Dr. Manuel de Rivas Cheriff, and Dr. H. Fernández Isassi. The Society is collecting a library of literature on the Conservation of Vision, and requests contributions or reprints from all physicians who are able to help in this movement.

The Pan-American Congress of Ophthalmology. As has been published in other issues of this Journal, the second Pan-American Congress of Ophthalmology will be held in Montevideo, Uruguay, from the 4th to the 10th of November, 1943.

The local committee is composed of the following physicians: president, Dr. A. Vasquez Barriere; vice-presidents, Drs. Carlos A. Berro and Washington Isola; treasurer, Dr. Julio A. Sicardi; secretaries, Drs. A. Paiva, R. Rodriguez Barrios, and C. Garbino.

The names of those constituting the program committee have been published in the April issue of the Journal, as well as the outline of the program.

Subscriptions may be sent to the secretary of the local committee, Dr. C. Garbino, Agraciada 3354, Montevideo, Uruguay.

Invitations to take part in the Second Pan-American Congress of Ophthalmology have been sent to all ophthalmologists in this hemisphere. However, as the list of oculists is far from complete, the directors of the Congress wish to extend a most cordial invitation through this Jour-

nal and through those who have received invitations, to all the eye specialists in the Americas to attend and read papers at the Congress at Montevideo. Invitations have also been sent to oculists in the mother countries of the American nations: Spain, Great Britain and her Empire, and Portugal.

In order to overcome the language difficulty, those who are to read papers and those who are to open the discussions will prepare a comprehensive résumé of their papers which will then be translated into the two other official languages of the Pan-American Congress of Ophthalmology (Spanish, English, and Portuguese). Slides will be projected during the presentation of the paper so that everyone present will be able to follow and understand the speaker. This method was used in Cleveland at the first meeting of the Congress with excellent results.

Societies

The Mexican Society of Ophthalmology and Otolaryngology has elected a new Board of Directors for 1943, as follows: president, Dr. J. Vicente Manero; vice-president, Dr. H. Fernandez Isassi; annual secretary, Dr. Carlos Tapia Acuna; treasurer, Dr. Pedro Berruecos Tellez.

The Brazilian Society of Ophthalmology has elected the following officers to serve during 1943-44: president, Dr. Ruy Rolim; first vice-president, Dr. Paiva Gonçalves; second vice-president, Dr. Jonas Arruda; secretary, Dr. Lincoln Caire; treasurer, Dr. Jose Alves Ferreira.

The Argentinian Ophthalmological Society held a theoretical-practical course on strabismus, under the supervision of Dr. Jorge Malbran. The course consisted of a series of five lectures held on December 7th, 9th, 10th, 11th, and 12th, at the Argentine Medical Association, and three surgical demonstrations held in the Santa Lucia Ophthalmic Hospital on the 8th, 10th, and 12th of the same month. The subjects included in the course were as follows: Mechanics of ocular movements; Binocular vision; Fusion; Disturbances of muscular equilibrium; Latent strabismus or heterophorias; Vision of the patient with strabismus; Etiopathogeny of strabismus; Convergent and divergent strabismus; Oblique strabismus; Treatment of strabismus; Paralyses of the extrinsic muscles of the eye; Paralyses of the third pair; and Supranuclear paralyses.

Personals

Canada. Dr. C. A. Campbell, of Toronto, is Canada's delegate to the Council of the Pan-American Congress of Ophthalmology in place of the late Dr. S. Hanford McKee.

Haiti. Dr. Elie Villard of the L'Hopital Général de Port-au-Prince is the Haitian delegate to the Council of the Pan-American Congress of Ophthalmology.

NEWS ITEMS

Edited by Dr. Ralph H. Miller
803 Carew Tower, Cincinnati

News items should reach the editor by the twelfth of the month

Deaths

Dr. John G. Steele, Bristol, Pennsylvania, died January 27, 1943, aged 68 years.

Dr. Lorenzo Nelson Grosvenor, Huron, South Dakota, died recently, aged 74 years.

Dr. Willard G. Mengel, Camden, New Jersey, died January 7, 1943, aged 46 years.

Dr. Michael B. Kelly, Wheeling, West Virginia, died December 10, 1942, aged 70 years.

Dr. Charles F. Yerger, Chicago, Illinois, died March 16, 1943, aged 63 years.

Dr. Elwood T. Easton, Boston, Massachusetts, died January 31, 1943, aged 66 years.

Dr. Carl B. Wagner, Chicago, Illinois, died February 1, 1943, aged 66 years.

Dr. William E. Boozan, Elizabeth, New Jersey, died February 5, 1943, aged 65 years.

Dr. Charles H. Boyer, Easton, Pennsylvania, died January 13, 1943, aged 73 years.

Dr. Frank A. Hartley, Springfield, Ohio, died January 18, 1943, aged 72 years.

Dr. Victor K. Martin, Buffalo, New York, died February 2, 1943, aged 48 years.

Dr. Clifton M. Miller, Richmond, Virginia, died February 26, 1943, aged 69 years.

Dr. Julius J. Buel, Lakewood, Ohio, died January 18, 1943, aged 82 years.

Miscellaneous

The American Board of Ophthalmology has announced that 1943 examinations will be held in New York City on June 4th and 5th, and in Chicago on October 8th and 9th. Candidates will be required to appear for examination on two successive days. Application blanks may be obtained from Dr. John Green, Secretary, 6830 Waterman Avenue, Saint Louis, Missouri.

A seminar in basic and clinical ophthalmology

was conducted at the Long Island College of Medicine, March 15th to 20th, for the purpose of supplying instruction to ophthalmologists interested in taking the examinations of the American Board of Ophthalmology. The following courses were given: "Embryology," by Dr. George H. Paff; "Clinical aspects of developmental defects," by Dr. Ralph I. Lloyd; "Physiology of vision," by Dr. Joseph Mandelbaum; "Clinical aspects of chemistry of the eye," by Dr. Joseph LoPresti; and "Applied perimetry," by Dr. John N. Evans.

The National Society for the Prevention of Blindness's program for the prevention of blindness from glaucoma is making headway. A demonstration glaucoma clinic has been organized at Manhattan Eye, Ear, and Throat Hospital, New York City. Volunteers who were trained to carry on certain phases of examinations of glaucoma patients are assisting in this clinic under the close supervision of ophthalmologists. Through the employment of a trained perimetrist for this clinic, the National Society for the Prevention of Blindness is offering similar training to five additional volunteers recommended for such training by chiefs of eye clinics of three hospitals in New York City.

Societies

The Brooklyn Ophthalmological Society held its regular meeting on April 15th. Participants in the scientific program were: Dr. Daniel B. Kirby, who presented a paper on "A system of intracapsular cataract extraction" illustrated with movies; and Dr. George H. Paff, who spoke on "Embryology of the human eye," a three dimensional demonstration with animated movies.

The Iowa State Medical Society held its ninety-second annual session on April 29th and 30th. Among the Iowa physicians who took part in the program were: Dr. Earl C. Montgomery, Atlantic, who spoke on "Intraocular neuritis"; Dr. Frank H. Reuling, Waterloo, on "Glioma of the optic nerve"; and Dr. Kenneth C. Swan, Iowa City, on "Abnormal retinal correspondence."

The nineteenth meeting of the Reading Eye, Ear, Nose, and Throat Society was addressed by Dr. Benjamin H. Shuster, of Philadelphia, on the subject, "Vertigo." Dr. Shuster brought out that vestibular tests ought to be interpreted in three classifications: 1. Normal responses. 2. Responses simulating the patient's symptoms. 3. Pathologic responses. He stated that ocular dizziness always disappears upon closing the eyes, and that labyrinthine dizziness is due to impaired water balance of the labyrinth unless it is due to inflammatory or degenerative pathology.

Personals

Dr. Otto Glasser of the Cleveland Clinic Foundation was the guest speaker at the March meeting of the Cleveland Ophthalmological Club. The subject of his lecture was "Present status of tests for color blindness."

Dr. Parker Heath, Detroit, spoke on "Traumatic eye injuries and epidemic keratoconjunctivitis" at the postgraduate industrial medical and surgical conference held at the Horace H. Rackham Memorial, Detroit, on April 8th.

Dr. William L. Benedict and Dr. Leonard M. Greenburg were recently appointed to the Board of Directors of the National Society for the Prevention of Blindness.

Dr. Paul Henry Case, Phoenix, Arizona, was among the speakers at the fifty-second annual meeting of the Arizona State Medical Association. He discussed "Recent advances in ophthalmology of interest to the general practitioner."

At the annual session of the Medical Association of the State of Alabama, Dr. Philip M. Lewis, Memphis, presented a paper, entitled "External diseases of the eye."

Among the speakers at the annual meeting of the Northern Tri-State Medical Association, held April 13th, was Dr. F. Bruce Fralick, who lectured on "Discussion and questions commonly asked about the eyes by patients."